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COMMUNICATIONS

THE LACRIMATION REFLEX*

BY

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ABERDEEN

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Introduction

IN the practice of ophthalmic surgery one of the most common complaints is that the eyes water, one of the rarest that the patient is unable to weep.

During the waking hours there is a constant flow of mucus and tears from the lacrimal and mucous glands. This secretion is distributed uniformly over the cornea by the blinking of the eyelids, and ensures adequate lubrication to maintain the brilliant polish on the main refracting surface of the eye. Normally very little secretion escapes through the canaliculi and lacrimal ducts, evaporation keeping pace with secretion.

Physiology

This normal secretion can be maintained by the accessory lacrimal and mucous glands alone, as has been demonstrated by von Graefe (1854) and others, who extirpated the main lacrimal glands without endangering the eye in any way from lack of lubrication. Cases suffering from keratoconjunctivitis sicca, reported by Sjogren (1940), had defective secretion from the accessory lacrimal glands as well as from the main lacrimal glands.

Apart from the flow of normal secretion, a flow of tears can be produced by (*a*) stimulating the terminations of the 5th nerve, especially those branches ending in the cornea and conjunctiva; (*b*) sneezing, coughing and vomiting; (*c*) psychic stimulation.

The irritation reflex is common to both man and animals, but psychic weeping is a phenomenon confined solely to humans. Numerous animals have been said to weep, but on investigation this has been proved to be a myth (Collins, 1932).

In human beings the flow of tears is exceedingly variable. Babies do not shed tears until they are a few weeks old; they howl, grow red in the face, screw their lids tightly together, but there is no flow of tears. Some women are exceedingly emotional and can weep on the slightest provocation, while it is exceedingly rare to see an adult male weep.

Nerve Supply

For the proper functioning of the lacrimation reflexes three nerves are said to be required. They are the 5th nerve, the 7th nerve, and the cervical sympathetic outflow. In addition, at least three ganglia take a part; they are the ganglion of the facial nerve (geniculate), the spheno-palatine, and the trigeminal ganglion (Gasserian). As the pathways and connections of these nerves and ganglia are very complicated, a description is added.

The lacrimal nerve.—This nerve is the smallest terminal branch of the ophthalmic division of the 5th nerve. It commences

in the fore-part of the cavernous sinus, and enters the orbit through the lateral part of the superior orbital fissure. It enters the lateral part of the fissure superior to the muscles and embedded in the periosteum. It then runs along the lateral wall of the orbit, superior to the lateral rectus and lateral to the lacrimal artery. Just before reaching the gland the nerve divides into two branches. The superior branch enters the gland, a few of its fibres ending there and supplying the secretory cells of the acini (Dogiel, 1893).

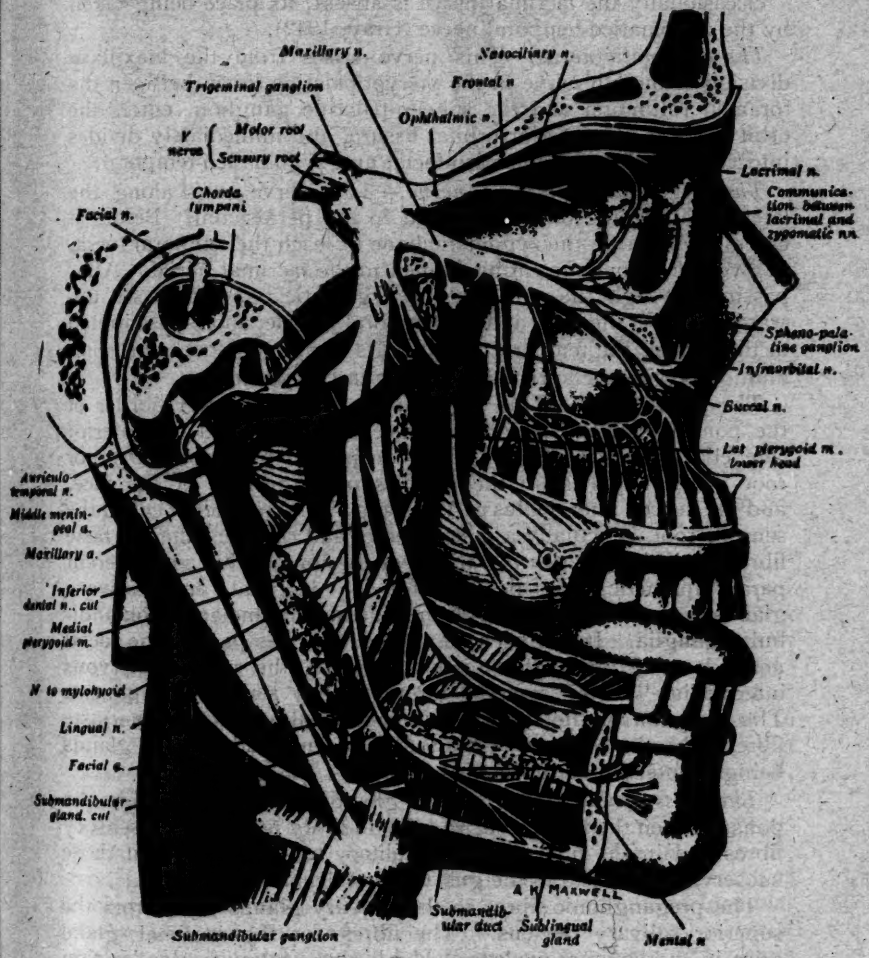

FIG. 1.

The right 5th nerve. (From Gray's Anatomy).

The majority of the fibres traverse the gland, pierce the septum orbitale, and end in the conjunctiva and the skin of the upper eyelid. Its peripheral terminations on the face join with filaments of the facial nerve. The inferior branch also enters the gland, but before doing so it gives off a branch which passes downward between the gland and the lateral orbital wall, and communicates with the zygomatico-temporal branch of the maxillary nerve (Fig. 1).

Occasionally the lacrimal nerve is absent, its place being taken by the zygomatico-temporal nerve (Gray, 1942).

The zygomatic nerve.—This nerve arises from the maxillary division of the 5th nerve in the pterygopalatine fossa, between the foramen rotundum and the spheno-palatine ganglion, enters the orbit through the inferior orbital fissure, and immediately divides into two branches, zygomatico-facial and zygomatico-temporal.

The zygomatico-temporal nerve.—This nerve runs along the lateral wall of the orbit embedded in the periosteum. Before it enters the canal in the zygomatic bone to reach the temporal fossa, it gives off a communicating branch to the lacrimal nerve. After passing through the canal in the zygomatic bone it passes upwards between the bone and the temporal muscle, and is distributed to the skin of the temporal region. Its terminal distribution communicates with the auriculo-temporal and facial nerves.

The facial nerve.—This nerve arises from the lower border of the pons lateral to the recess between the olive and the inferior cerebellar peduncle (Fig. 2). It consists of two roots, a motor root which supplies all the muscles of expression of the face, and the nervus intermedius of Wrisberg. This root, often called the sensory root, is actually a mixed nerve, and contains afferent taste fibres from the anterior two-thirds of the tongue, but also efferent parasympathetic fibres destined for the lacrimal and salivary glands by connections through the spheno-palatine and submandibular ganglia. It has been suggested that this part of the facial nerve be called the glosso-palatine nerve, the name "nervous intermedius" being reserved for the purely sensory component. The ganglion of the facial nerve is the cell station for the taste fibres only, the secretory fibres to the lacrimal and salivary glands being uninterrupted in this ganglion.

The glosso-palatine nerve.—The glosso-palatine nerve enters the pons between the motor root and the auditory nerve. The sensory fibres end in the nucleus of the solitary tract (Fig. 2), but those subserving taste end in the gustatory nucleus.

The preganglionic fibres to the salivary glands come from the superior salivary nucleus. The fibres for the lacrimal gland arise in the lacrimal nucleus. The higher centres for lacrimation are quite unknown.

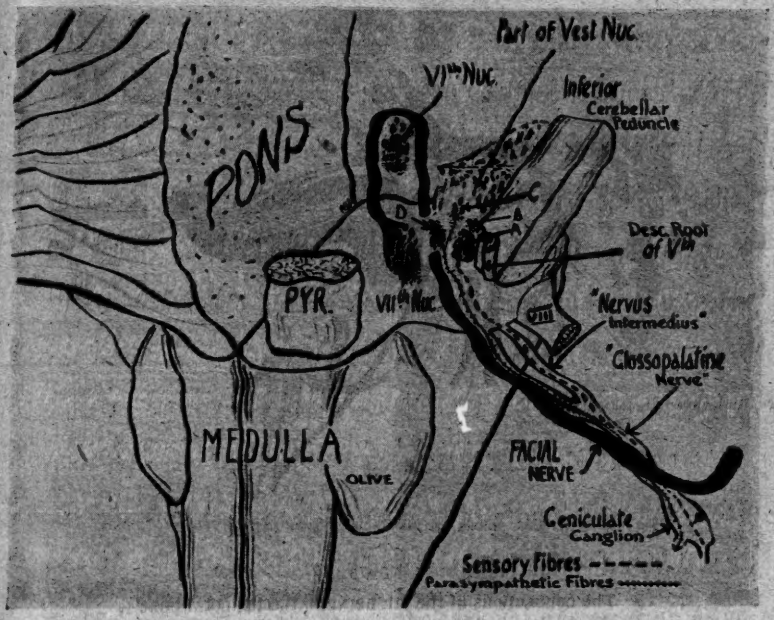

FIG. 2.

The 7th nerve and its central connections. The sensory fibres end in A (the nucleus of the solitary tract) but those subserving taste may end in B (the so called "gustatory nucleus"). The preganglionic fibres to the salivary glands come from C (the superior salivary nucleus). Those for the lacrimal gland may come from this nucleus, but some believe that they arise in D (which is then called the "lacrimal nucleus").

After the facial nerve enters the internal auditory meatus it passes outwards and forwards (Fig. 3). It then makes nearly a right-angled turn, the genu. From this point it proceeds backwards and slightly downwards to its exit at the stylo-mastoid foramen. In the facial canal, where the nerve bends sharply to form the genu, there is an oval swelling on the nerve, the *facial (geniculate) ganglion*. From the ganglion three small nerves arise, (*a*) *The greater superficial petrosal nerve*. This nerve passes forwards through the hiatus on the anterior surface of the petrous portion of the temporal bone, and enters a groove in the bone, covered by the dura mater. Before leaving the bone it receives a twig from the tympanic plexus. It passes deep to the trigeminal (Gasserian) ganglion and reaches the foramen lacerum, where it is joined by the deep petrosal nerve from the sympathetic plexus on the internal carotid artery to form the nerve of the pterygoid

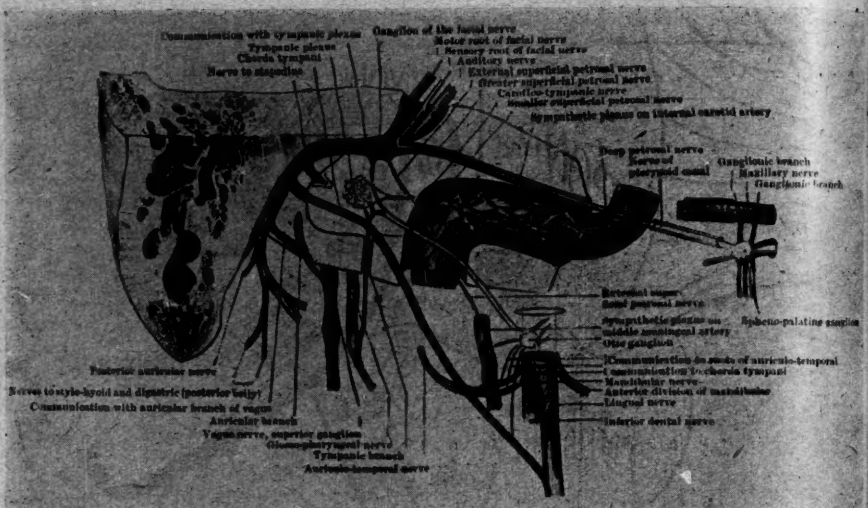

FIG. 855.—THE CONNEXIONS OF THE FACIAL NERVE IN THE TEMPORAL BONE.

FIG. 3.

The connections of the 7th nerve in the temporal bone. (From Cunningham's Anatomy).

canal or Vidian nerve, which passes forwards through the pterygoid canal and ends in the spheno-palatine ganglion. (*b*) A small branch runs in the bone to join with the tympanic branch of the glossopharyngeal, to form the *lesser superficial petrosal nerve* (Figs. 3 and 5). It is conveyed through this nerve to the otic ganglion. (*c*) *The external petrosal nerve* is an inconstant branch which connects the ganglion with the sympathetic plexus on the middle meningeal artery.

In the descending part of the canal three branches leave the facial nerve: (*a*) The nerve to the stapedius; (*b*) the chorda tympani. It arises from the facial nerve 6 mm. above the stylo-mastoid foramen and supplies the parasympathetic root to the submandibular ganglion, and taste fibres to the anterior two-thirds of the tongue. (*c*) Just before the nerve reaches the stylo-mastoid foramen a branch is given off which communicates with the auricular branch of the vagus.

The spheno-palatine ganglion and its connections.—The spheno-palatine ganglion lies in the upper part of the pterygo-palatine fossa, close to the spheno-palatine foramen (Fig. 1). It is a small reddish-grey body situated below the maxillary nerve. It receives three kinds of fibres, sensory by the spheno-palatine branches

from the maxillary nerve, and sympathetic and parasympathetic from the nerve of the pterygoid canal, the former coming from the carotid plexus by the greater superficial petrosal nerve.

The branches of the spheno-palatine ganglion are divisible into four groups—orbital, palatine, nasal and pharyngeal. Meantime only the first group concerns us. The orbital branches consist of two or three filaments which enter the orbit through the inferior orbital fissure and are distributed to the periosteum, the orbitalis muscle, to the optic nerve and sheath and, according to Cunningham, to the lacrimal gland. Only those fibres that are destined for the lacrimal gland are relayed in the ganglion.

The sympathetic pathway—the deep petrosal nerve.—This nerve arises from the cervical sympathetic plexus. It runs in the carotid canal lateral to the internal carotid artery. In the substance of the fibro-cartilage that fills the foramen lacerum, it joins with the greater superficial petrosal nerve to form the nerve of the pterygoid canal or Vidian nerve.

The important features of the anatomy are :—

1. The lacrimal nerve communicates with the zygomatico-temporal branch of the maxillary nerve and the terminal fibres of the 7th nerve, but ends chiefly in the conjunctiva and skin of the upper eyelid. Both branches traverse the lacrimal gland, and send filaments to the acini.

2. The zygomatic nerve leaves the maxillary nerve at an acute

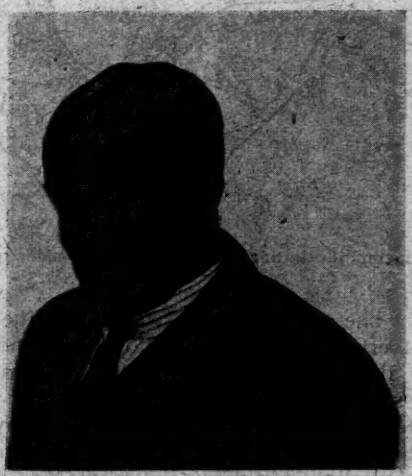

FIG. 4.

The connections of the 7th nerve in the temporal bone.

angle midway between the spheno-palatine ganglion and the trigeminal (Gasserian) ganglion (Fig. 1) and all the fibres can be traced into the latter ganglion. It would appear, therefore, that lacrimation impulses traversing the spheno-palatine ganglion must first pass to the trigeminal (Gasserian) ganglion along the maxillary nerve before they can enter the zygomatic nerve and pass along its zygomatico-temporal branch (Fig. 4).

3. Para-sympathetic fibres to the lacrimal and sub-lingual glands accompany each other in the glossopalatine nerve (nervus

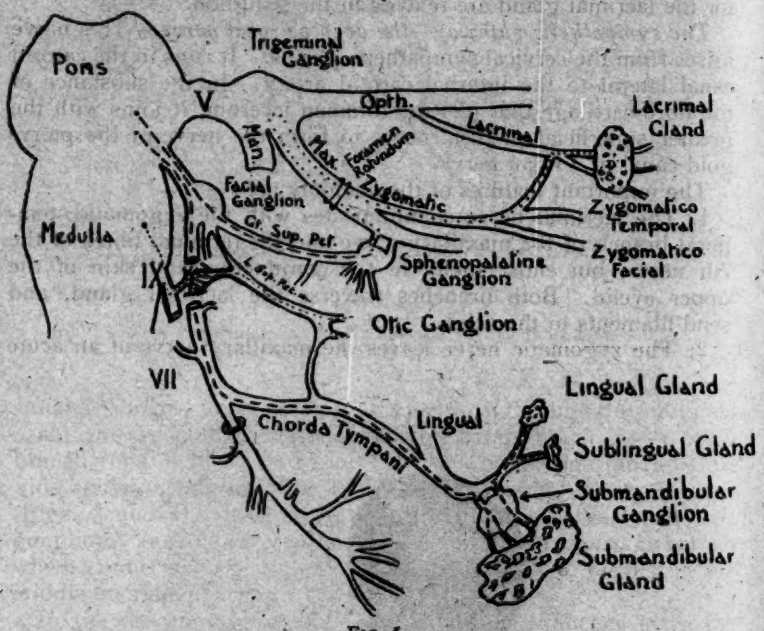

FIG. 5.

Diagram illustrating the syndrome of crocodile tears.

intermedius). The pre-ganglionic pathway for the former is along the greater superficial petrosal nerve to the spheno-palatine ganglion, and to the latter along the chorda-tympani and lingual nerves to the submandibular ganglion (Fig. 5).

These anatomical details have been verified by dissecting six still-born babies. Slight modifications have been seen, such as the lacrimal being a branch of the frontal nerve, the lacrimal communicating with the frontal nerve, and in one case with the ciliary ganglion.

Clinical and Experimental Observations

A considerable amount of clinical and experimental evidence can be found to support the view that impulses for lacrimation actually pass along these nerves, and by examining patients who have had the various nerves sectioned by operation, the actual part taken by these nerves in the different reflexes can be surmised. As the lacrimal nerve traverses the lacrimal gland besides terminating in the conjunctiva and skin of the upper eyelid, the first point to be settled is whether efferent as well as afferent impulses run in this nerve. Parsons (1902) stimulated the peripheral cut end of the lacrimal nerve in the cat, dog and rabbit, and in no case did tears run down the face, nor was any activity shown microscopically in the gland. It would appear, therefore, that the fibres that terminate in the gland are those that come along the branch communicating with the zygomatico-temporal nerve, or from branches that pass directly from the spheno-palatine ganglion into the orbit.

Evidence that the 5th nerve is the afferent pathway for lacrimation caused by irritation of the eye and conjunctiva is fairly conclusive.

Experimental

Effect of tear gas upon the normal and anaesthetic eye.—The left eye of a young normal subject was occluded with a pad and bandage and the nostrils were plugged with cotton-wool. The right eye was then anaesthetised by means of several drops of 4 per cent. cocaine. Tear gas* was then directed on to the right eye by means of a scent spray. No pain was felt in the eye and there was no lacrimation. The pad and bandage were then removed and the gas was projected on to both eyes. Great pain was felt in and around the left eye, and there was profuse lacrimation from this eye only.

Effect of foreign body upon cornea of eye.—A small piece of grit was placed on the cornea of a normal eye. There was great pain in the eye and profuse lacrimation from this eye only.

Lacrimation is, therefore, initiated by stimulation of the nerve endings in the cornea and conjunctiva and is unilateral.

Clinical Investigations

Foreign body on cornea.—A foreign body had to be removed from a child's eye, so the eye was rendered anaesthetic by means of 2 per cent. cocaine. The child was frightened and wept, a profuse flow of tears taking place from both eyes as the result of psychical stimulation.

* Phenyl-bromo-aceto-nitrite. (Supplied by the War Office).

Toxic neuritis of the ophthalmic division of the 5th nerve.—A male, age 55 years, consulted me because his right eye had turned dim a few days previously. There was no pain, but the eye watered a good deal. Upon examination it was found that vision was reduced to counting fingers at 3ft. The eye was red, the cornea was hazy and covered with superficial dot-like opacities. The tension was normal. The cornea, eyelids, forehead, and right side of nose were found to be anaesthetic. There was no sign of herpes. His teeth were very bad, and pus was oozing from the gums. Toxic neuritis of the ophthalmic division of the 5th nerve was diagnosed. The teeth were removed, and the eye slowly recovered over a period of six months, but poor vision remained owing to corneal nebulae. During treatment there was profuse lacrimation from the right eye only, but pain was never present. As the cornea and eyelids were completely anaesthetic to touch and pain, the stimulus for the excessive lacrimation could not have originated in the conjunctiva or cornea, and as the lacrimation was unilateral it was not psychical in origin.

Herpes ophthalmicus in a woman aged 65 years.—The cornea became involved but cleared up well under treatment. The cornea was still anaesthetic six months after the appearance of vesicles. The patient was rather emotional and wept fairly readily, tears appearing equally in both eyes. In this case no undue lacrimation was observed during the acute phase, No interference, however, was present with the flow of tears from psychical stimulation.

Post-operative 7th nerve paralysis.—A woman, age 47 years, was admitted into the Ear, Nose and Throat ward of the Aberdeen Royal Infirmary on March 17, 1937, suffering from acute mastoid disease, and underwent operation upon the day of admission.

Immediately following operation it was noticed that she had a left facial paralysis peripheral in type. The skin of the forehead was flattened, she was unable to close the left eye, and the side of the mouth drooped.

For the next few weeks there was troublesome watering from the left eye and there was loss of taste on the left side of the tongue over the anterior two-thirds.

On September 22, 1937, there was still slight weakness of the left side of the face. The eyelids were in good position and it was possible to close the left eye voluntarily, although not so tightly as the right. She complained of slight pain over the supra-orbital notch. She still complained of watering of the left eye. The patient was very emotional during her illness and wept readily, tears flowing equally from both eyes. In this case, of course, there was no sensory loss in the eyes or eyelids. There was excessive lacrimation during the time of the paralysis, and the flow of tears on emotional occasions was normal, so both the

afferent as well as the efferent pathways were intact. As there was a loss of taste on the anterior two-thirds of the tongue, the lesion of the facial nerve must have been central to the point where the chorda-tympani nerve leaves it. If we assume that lacrimation impulses pass along the greater superficial petrosal nerve, the lesion must have been between where the chorda-tympani nerve leaves the 7th nerve and the facial (geniculate) ganglion (Fig. 3).

Mixed-celled sarcoma invading the base of the skull.—A woman, age 54 years, was admitted into Ward 8, Aberdeen Royal Infirmary, on April 9, 1936. She complained of pain and swelling of the left face and neck of six months' duration. The pain varied in intensity, being occasionally very acute, and extended from above the frontal eminence to below the angle of the jaw, and from behind the ear to the mid line of the face. There was no discharge from the left ear. The swelling in front of the ear did not increase in size before or during meals, and there was no loss of taste or undue dryness of the mouth.

Upon examination there was a large, hard, fixed swelling in front of the left ear. The swelling filled up the angle of the jaw and extended forwards on the cheek to near the side of the nose and angle of the mouth. The swelling also extended to behind the ear.

There were no palpable lymph nodes in the neck or axilla. There was an area of partial anaesthesia corresponding to the radiation of pain. There was no facial paralysis and the corneal reflex was present.

The swelling was explored with a needle, but no fluid was found. A piece of the tumour was then removed for biopsy, and upon examination it was found to be a mixed-celled sarcoma.

On September 19, 1936, a radium plaque was applied over the swelling and left in position for 80 hours.

The patient was discharged on September 30, 1936, free from pain.

On February 2, 1937, she was re-admitted to hospital. The patient said that shortly after she was discharged from hospital the pain again commenced, at times being very severe. It radiated over left cheek, side of head, nose, lips and chin as far as the mid-line. Upon examination it was found that the tumour had increased in size. There was still no facial paralysis and the corneal reflex was still present.

On February 8, 1937, the trigeminal (Gasserian) ganglion was exposed by the temporal route, and the sensory root of the 5th nerve was cut. No attempt was made to save the motor root. Tumour tissue was seen to be infiltrating the floor of the skull in the neighbourhood of the foramen ovale. Considering the

situation and extent of the tumour, it was thought that the sphenopalatine ganglion was possibly destroyed. To expose the trigeminal (Gasserian) ganglion the greater superficial petrosal nerve was cut.

During the following week the patient was mentally confused, but did not complain of pain. She was very emotional and wept readily, tears flowing from the right side only.

The tumour steadily increased in size, tumour tissue being occasionally discharged from the nose. The operation scar and soft palate became infiltrated, and she died on April 25, 1937.

Section of the sensory root of the 5th nerve.—A male, age 56 years, suffered from trigeminal neuralgia for many years. The sensory root of the 5th nerve was sectioned by the temporal route. The area as shown (Fig. 4) was completely anaesthetic to pain, touch, heat and cold. Four years after operation the eye was quiet, vision was 6/6. Neuro-paralytic keratitis had not at any time been present. The right eye was occluded with a pad and bandage, and tear gas was directed on to the left eye. There was no lacrimation. The pad and bandage was then removed and the gas was directed on to both eyes: There was pain in and around the right eye only, and tears ran down the cheek from the right eye. This patient was an actor, and he was asked to take particular notice if tears flowed from the left eye, if at any time he wept from any emotional cause. After a six months' interval he reported that tears flowed on emotional occasions from the right eye only.

Section of the sensory root of the 5th nerve has now been performed for many years for the relief of trigeminal neuralgia. In performing the operation by the temporal route, some surgeons make the approach to the trigeminal (Gasserian) ganglion by stripping the dura from the base of the skull (Rowbotham, 1939). In elevating the dura the greater superficial petrosal nerve may be torn or cut in the process. Some surgeons deliberately cut the nerve, as it is thought that it was by traction on the greater superficial petrosal nerve that the 7th nerve was injured in the Fallopian canal, causing transient facial paralysis after the operation. Some surgeons again approach the ganglion intradurally. By this method of approach any injury to the greater superficial nerve is prevented. It is interesting to note that this means of approach was advocated by Lockhart (1925) upon anatomical grounds, as being the easiest method of approach to the ganglion. When this method of approach to the ganglion is used no diminution of lacrimation takes place (Rowbotham, 1939).

Dandy (1929) approached the sensory root of the 5th nerve from the posterior fossa, and the root was sectioned just before it entered the pons. In all of his cases tears flowed equally from both eyes on emotional occasions.

By sectioning the sensory root of the 5th nerve no reflex lacrimation takes place owing to the break in the afferent pathway. If no other nerve is interfered with, emotional weeping should, however, still take place normally, as the stimulus probably originates in the cerebrum. When the operation is performed by the temporal route instead of by the posterior fossa route, the only additional nerve to be divided is the greater superficial petrosal. As the former operation interferes with weeping while the latter does not, motor impulses to the lacrimal gland must pass along the greater superficial petrosal nerve.

Herpes of the geniculate ganglion.—Patient is a male student, age 23 years. Four years ago, while he was walking along the street, he felt something wrong with the right side of his face, and when he reached home and looked in the mirror he saw that there was a complete paralysis of the right face. He was unable to close the right eye, and he was unable to whistle. During the evening he developed a temperature and was admitted to hospital. On examination it was seen that he had a flaccid paralysis of all the muscles of the right side of the face. The paralysis was at a maximum from the first. There was no rash on the face or ear, but there was a rash on the roof of the mouth well forward. There was no loss of touch sensation on the face or tongue, but sense of taste was lost on the anterior two-thirds of the tongue on the right side. Hyperpyrexia continued for four days, the maximum being 101°F. During his stay in hospital nothing distinctive about lacrimal secretion was observed.

Upon examination four years later partial paralysis of the right side of the face was found to be still present. The forehead was smooth, the right eye could only be partly closed, the mouth drooped at the corner, and he was unable to whistle. When he attempted to use one group of muscles, as in winking, the muscles of the mouth were also put into action. When he ate he did not weep.

Upon testing for lacrimal secretion with the blotting-paper test, both eyes were found to be equal. This case was diagnosed as herpes zoster of the geniculate ganglion. As there was a complete paralysis of the motor component of the 7th nerve along with the absence of taste on the anterior two-thirds of the tongue, one would have expected that there would have been some interference with lacrimation. While he was in hospital no special notice was taken about lacrimation, and no test was made to see whether or not it was absent. Upon questioning the patient four years afterwards, it appears to me significant, however, that during the time he was in hospital and when he subsequently got outside, he was not troubled with the eye watering, as invariably happens when the paralysis is peripheral to the geniculate ganglion. When

I examined him four years later a certain amount of power had returned to the facial muscles, so it is probable that the nerve to the lacrimal gland had also regained its function.

Syndrome of crocodile tears.—A male, age 60 years, came to the out-patient department, Aberdeen Royal Infirmary, with a complaint that when he ate tears overflowed from his right eye and water ran from his right nostril.

He gave a history of a sudden right-sided facial paralysis six months previously. The lacrimation commenced with the return of power to the face three months after the paralysis. Tears flowed most readily when hot tea or soup was taken. Thinking about food did not cause lacrimation. He did not complain of loss of taste, but he said there was a different feeling in the right side of the mouth from the left, and that he had a hot feeling below the right eye and a tight feeling in his cheek. He can now wrinkle his forehead and close his right eye. The mouth is slightly twisted and he is unable to whistle. When he wrinkles his forehead all the muscles of the right side of the face fibrillate. He is deaf on the right side.

When he was given a plate of soup, tears ran down his face on the right side and he had to blow his nose frequently.

Such a syndrome can only be produced by a lesion of the 7th nerve central to the facial ganglion, as the nerves divide at the ganglion (Fig. 5).

Ford (1933) published three cases of this condition and Savin (1939) described other three. For the relief of the condition the spheno-palatine ganglion was injected with alcohol through the nose by Gottesfeld and Leavitt (1942).

In his comment Ford maintains that the contracture is due to increased tonus of the muscles and not to fibrous shortening of the muscles. The contracture is not always present to the same degree, but is increased by any form of animation, and is at a minimum when the patient is relaxed. When it is desired to activate any single group of muscles so as to produce a simple action, such as blinking the eyelid, all the muscles of the side of the face are brought into action.

It is supposed that the regenerated fibres have become fused and misdirected, and instead of a single muscle being stimulated, all the muscles of the face are stimulated indiscriminately. As fibres to the lacrimal and salivary glands also run in the 7th nerve, misdirection of these regenerating fibres also takes place, so that the patient lacrimates when he salivates. It is interesting to note that lacrimation could be stimulated only by taking food into the mouth, and not by bringing into action any of the facial muscles.

No one has described the associated flow of water from the nostril of the same side. Two explanations are possible: (1) The flow

of water could come from the eye via the naso-lacrimal duct. (2) The nerve supply to the mucous membrane of the nose comes from the spheno-palatine ganglion via the palatine nerves.

It is possible, therefore, that secretory fibres to the nose accompany the secretory fibres to the lacrimal gland as far as the spheno-palatine ganglion.

Foville's syndrome.—A male, age 17 years, complained of double vision, and the following day he developed a left facial paralysis. He had no recent illness, no headache, but was actively sick on two occasions upon the day that diplopia occurred. He was very giddy and unable to walk straight.

Upon examination it was seen that he had a left facial paralysis of all the muscles of expression. He could, however, close the left eye.

Vision was 6/6 both eyes, and accommodation was normal for his age.

The left pupil was slightly larger than the right, but both reacted normally to light.

When vision was directed straight ahead there was no squint. Movement of either eye to the left was impossible.

Movement of the right eye to the right was restricted and was accompanied by a rough horizontal nystagmus. Movement of the left eye to the right was limited to about 5 degrees. Upward and downward movements were full. Convergence also was full.

With the blotting-paper test lacrimation was increased upon the paralysed side.

The fundi were normal and the fields were full. Apart from a positive Romberg the physical examination was entirely negative. This included a complete neurological examination, X-ray of skull, examination of the cerebro-spinal fluid and blood, and a culture from a throat swab. He had not a raised temperature.

From the fourth day after the development of the facial paralysis recovery began to take place, and within a fortnight he had returned to normal.

In 1939 I reported another case of this syndrome (Mutch, 1939), but the defect was permanent and the paralysis of the eye movements was confined to the one side. Both, however, had unequal pupils, the smaller pupil being at the opposite side from the paralysis.

The lesion is situated in the pons near the floor of the 4th ventricle where the fibres of the 7th nerve sweep round the 6th nerve nucleus, and come in close relation with the medial longitudinal bundle (Fig. 6).

As there is no loss of lacrimation the fibres destined for the lacrimal gland must join the 7th nerve peripheral to the 6th nerve nucleus.

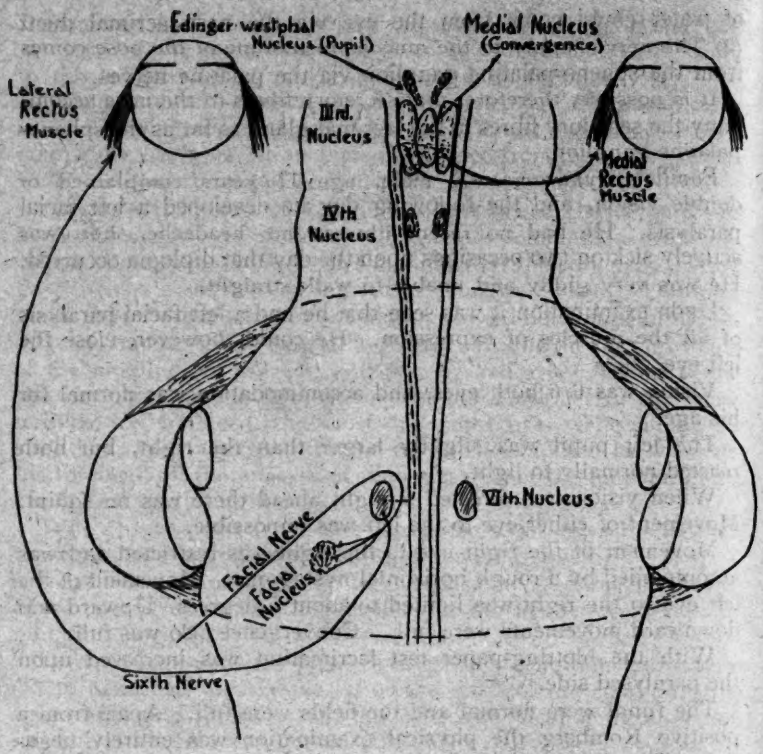

FIG. 6.

Diagram showing the nuclei, nerves, and tract involved in Foville's syndrome.

Congenital absence of weeping.—In 1930 Duke-Elder recorded two cases and reviewed the literature on the subject, and was able to find only three others. In no case was the absence of weeping associated with the absence of salivation. Duke-Elder in the same article also reviews the literature of recorded cases who developed loss of lacrimal secretion after birth. He divides these cases into two series: (1) Traumatic in origin, whereby the nerves to the lacrimal glands were injured by fracture of the skull, or the lacrimal glands were removed by operation. (2) Cases occurring spontaneously in women after the climacteric. In some cases the loss of lacrimation was associated with a dryness of the mouth and the skin. The majority, however, in all sections had an associated conjunctivitis or keratitis.

In 1940 Sjögren contributed to Modern Trends in Ophthalmology the section upon keratoconjunctivitis sicca. He states

that the condition is confined almost solely to women, and that a specific test for the condition is the instillation of a 1 per cent. watery solution of bengal red into the conjunctival sac. In positive cases the bulbar conjunctiva and cornea are stained a deep red over an area corresponding to the palpebral fissure.

Sjögren found xerostomia (dry mouth) present in ten out of twenty-two cases of kerato-conjunctivitis sicca, and associated arthritis in fourteen cases.

Congenital absence of weeping associated with absence of salivation.—Mrs. F., age 67 years. Height 4ft. 10in.

This old lady had never wept, and had suffered from defective secretion in her mouth all her life. While Mrs. F. was still an infant the family doctor noticed that when she cried no tears came. As the child grew older he learned that dry food could not be eaten, so came to the conclusion that salivation as well as lacrimation was defective. He even gave the girl a mixture to try to increase the flow of saliva and tears, but there was no response. The reaction to peeling onions was also negative. All her life she had had frequent attacks of indigestion, and had to be careful in the choice of food. While eating she took large quantities of liquid, and even between meals took frequent drinks of water. She had diphtheria, scarlet fever, and typhoid fever when young. She had worked hard all her life, and as her husband died five weeks after marriage she had to work to support herself.

She had no family, the menopause coming on at the age of 50. She had no complaint about her eyes. There was no undue dry feeling. Should a foreign body enter the eye the eye turned red, but there was no lacrimation. She had worn glasses for the correction of hypermetropia and presbyopia for many years. On examination the eyes were deep-set, with narrow palpebral fissures. The lids were in normal position. The puncta were present and patent. On everting the eyelids the conjunctiva was of healthy colour and moist. The corneae were clear, moist, and regular. The pupils were equal and active. There was no other eye abnormality present. Her doctor told me that he had looked at her mouth on several occasions and always found it dry. When I examined it her tongue and mucous membrane were dry but of good colour. She was given a sweet to suck, and two hours later the size of the sweet was very little reduced. She was then given a small cube of paraffin wax to chew and was instructed to spit any saliva that gathered in her mouth into a clean test-tube that was provided. At the end of three hours there was 2 c.c. of a mucoid secretion in the test-tube. It was analysed with the following result:—

1. Microscopic deposits. Squamous epithelial cells present. No crystals.

2. No thiocyanates present as usually found in saliva.
3. No ptyalin present.

This was compared with controls of normal saliva.

Congenital absence of 7th nerve.—A female child was born with a flaccid paralysis of all the muscles of one side of the face. From the appearance at birth it was assumed that there was a congenital absence of the 7th nerve on that side. The mother was asked to observe if tears flowed from both eyes. The child, although only beginning to speak, was very co-operative, and when she hurt herself she ran to her mother and said, "Look, tears, mummy." Tears flowed equally from both eyes.

Unilateral sympathetic ganglionectomy.—A female, age 19 years, was operated upon for the relief of Raynaud's disease, the sympathetic chain being cut below the stellate ganglion and again below the 3rd thoracic ganglion, and the intervening segment removed.

By saving the 1st thoracic white ramus communicans and the stellate ganglion, the sympathetic supply to the head and neck was left practically intact.

Several weeks after the operation, upon examination of the eyes, no Horner's syndrome was present on the operated side, and both pupils dilated equally when cocaine was instilled. Lacrimation was normal, tears due to irritation and to psychical stimulation appearing equally in both eyes.

Unilateral cervical sympathetic ganglionectomy.—A small boy, age 10 years, was operated upon for the relief of scleroderma, the stellate ganglion on one side being removed at operation. When he was examined one month after operation it was seen that he had a Horner's syndrome on the operated side, and the pupil failed to dilate with cocaine. During one experiment the boy's arm was pricked with a pin and he wept, tears running down his cheeks from both eyes.

Several other patients, both male and female (who had had unilateral ganglionectomy performed), were examined at various periods after operation, and on no occasion could any difference be seen in lacrimation or would the patients admit that there was any diminution or alteration in lacrimal secretion following operation.

Bilateral sympathetic ganglionectomy.—A girl, age 22 years, was operated upon three years previously for the relief of Raynaud's disease, bilateral cervical sympathetic ganglionectomy being performed, the stellate ganglion being removed at operation.

The patient had a bilateral Horner's syndrome. Upon examination the eyes appeared normal in every way, vision was 6/6, the fields were full, the pupils reacted normally to light and near vision, and the fundi were normal. The pupils failed, however,

to dilate upon the instillation of 4 per cent. cocaine. The patient complained that towards night the eyes felt small and uncomfortably dry. Upon weeping tears flowed normally from the eyes. Lacrimation was also profuse during vomiting and when a foreign body entered an eye.

Conclusions

Reflex lacrimation is unilateral and can be inhibited by surface anaesthesia of the conjunctiva and cornea, and by section or paralysis of the ophthalmic division of the 5th nerve. This nerve is, therefore, the sensory or afferent pathway for reflex lacrimation.

The efferent pathway runs in the 7th nerve, but is independent of the motor fibres to the face muscles (syndrome of crocodile tears), and is separate from the 7th nerve in the brain near the 6th nucleus (Foville's syndrome) and also peripheral to the geniculate ganglion (Bell's palsy with loss of taste on anterior two-thirds of tongue).

If the greater superficial petrosal nerve be cut or the spheno-palatine ganglion be blocked no reflex or psychical lacrimation takes place, even although the ophthalmic division of the 5th nerve is left intact, while psychical lacrimation is normal if the sensory root of the 5th nerve be cut and the greater superficial petrosal nerve spared. Motor impulses must therefore pass along the greater superficial petrosal nerve.

Psychical weeping is bilateral and cannot be inhibited by surface anaesthesia or paralysis of the 5th nerve or section of the cervical sympathetic nerve.

Upon anatomical grounds it is assumed that the fibres from the spheno-palatine ganglion which enter the maxillary nerve run to the trigeminal (Gasserian) ganglion before they pass along the zygomatic nerve.

No interference with reflex or psychical lacrimation results from either unilateral or bilateral section of the cervical sympathetic chain in the neck. One patient did complain that her eyes felt unduly dry towards night, so there is a possibility that mucus from the glands in the conjunctiva may be inhibited, but on no occasion has keratitis sicca been observed in any patient who has had a sympathectomy done. It is unlikely, therefore, that the sympathetic nerve takes any part in reflex or psychical lacrimation.

I wish to express my gratitude to Professor J. R. Learmonth for his help and advice in planning this investigation.

For allowing me to investigate and report upon their cases I also tender thanks to :—

Professor J. R. Learmonth, cases 5, 6, 7, 10, 12, 13, 14; Mr. J. Otty, case 4; Dr. B. Law, case 8; Dr. G. D. Roche, case 11.

Thanks are also due to Professor R. D. Lockhart and his assistants in the Anatomy Department for the criticism and help they gave with the anatomy and diagrams. Also to Dr. A. Lyall for the examination of saliva.

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VITAMIN P IN OPHTHALMOLOGY

BY

W. R. MATHEWSON

EDINBURGH

IN two cases of ocular haemorrhage recently under treatment vitamin P was used with very satisfactory results.

In the first case the patient had very extensive retinal haemorrhages and nasal and bladder haemorrhages, but on giving vitamin P the nasal and bladder haemorrhages ceased, no fresh retinal haemorrhages occurred, and those present absorbed. He suffered from myelomata, and the nasal haemorrhage was characterised by large clots of blood and serum.

In the second case the patient had recurrent haemorrhage into the anterior chamber after extraction of a cataract, but on giving vitamin P there was no recurrence, and the iris, which had become muddy, rapidly cleared. The eye, which was looking very unhealthy, rapidly took on a healthy tone. The patient improved greatly and rapidly in general appearance and mental agility when vitamin P was given.

FIRST CASE—General History

A University graduate of high intellectual attainment and regular life was first seen in 1935 at the age of 47, and thereafter from time to time for refractive errors and the like.

* Received for publication, February 14, 1944

In July, 1943, he fell very ill from multiple myeloma, and for this condition he received X-ray " spray treatment," with a warning of the possibility of petechial haemorrhages.

About this time he suffered from bladder and nasal haemorrhages, but no local causes for these were diagnosed, though he was in hospital for thorough investigation.

The prognosis was considered to be hopeless at the time when vitamin P was commenced.

Ophthalmological Notes

What follows is a condensation from several visits, some to his bedside, for he was for a time too weak and jaundiced and short of breath to get up.

Many observations, such as field examination, were omitted because he was very ill, and to avoid drawing the patient's attention to his condition.

Onset.—(1) About August 12 he found himself unable to see to read. (2) Some days later he got out of bed, early in the morning, to shave, and had a sudden onset of severe headache, and after this, so far as can be ascertained, his general condition and his vision became worse. He did not remember this incident, but it was thought to have been a subarachnoid haemorrhage. The headache was temporal and mainly on the left side.

First Ophthalmological Examination

August 15, 1943.—*Vision* with glasses was R.E. J.2, L.E. J.6. *Fundi*: The right eye had much vascular engorgement and many haemorrhages. There were haemorrhages on the disc and nerve fibre layer haemorrhages medial to the disc, small round feathery edged haemorrhages above the macula, and a large subhyaloid haemorrhage below the macula. Patches of exudate were also present. At the macula there was a stippled area almost a quarter disc in diameter and noted as " leathery edged," for its edge looked like leather.

The left eye showed more extensive haemorrhages, and the appearance suggested, at first sight, central venous thrombosis.

Treatment.—Vitamin P was prescribed at this time.

Further Ophthalmological Examination

August.—Towards the end of August it was noted that some of the patches of exudate appeared to be absorbing, but that the veins and arteries were in places almost indistinguishable except by using a red-free filter on the ophthalmoscope, that they appeared in places empty and in others filled with an almost clear or translucent pinkish fluid, and looked as if buried in an oedema overlying the retinal surface. There were no fresh haemorrhages.

The haemoglobin was down to 40 per cent. and the R.B.C. was about 2,000,000 at this time.

September.—Dr. A. H. Sinclair kindly saw the case in consultation and confirmed the retinal conditions, and agreed that so far as could be determined they were associated with retinal venous thrombosis and subarachnoid haemorrhage.

In the right eye there was altitudinal hemianopia which appeared to be associated with the subhyaloid haemorrhage and diminished as this haemorrhage absorbed.

Later in the month some right homonymous hemianopia was detected and was still present in some degree in November, and was at the time thought to be connected with subarachnoid haemorrhage. The hemianopia was detected because the patient stated that on his tin of glucose he could now, binocularly, read the word "Glucodin," whilst previously he could read only "Gluco."

The fundi were now clear enough for it to be seen that the haemorrhages and exudates were in many cases in the region of arterio-venous crossings, an important point, and at some points the tributary veins could clearly be seen to be threadlike and empty.

Almost immediately after the commencement of administration, in August, of vitamin P, nasal and bladder haemorrhages ceased, and also, it is emphasized, there were no further retinal haemorrhages.

In September, however, there was a short period during which vitamin P was omitted and nasal haemorrhage recommenced, and this was considered to be one of the indications that vitamin P was really having some therapeutic action.

October.—Early in October Dr. Scarborough kindly arranged for the capillary permeability to be ascertained by his negative pressure method and by his apparatus, and it was found to be about 450, the normal being about 300, and haemorrhages from capillaries, it is to be noted, are liable to occur under 250.

Haemoglobin now 64 per cent. and R.B.C. 2,760,000.

November.—Vitamin P had been commenced in August, 4 tablets of permidin (Glaxo) daily, and continued for six weeks, and then stopped early in October when the capillary permeability was 450. Early in November the capillary permeability was found to be below 250, after the vitamin P had been stopped for 20 days. Vitamin P administration was therefore recommenced, this time two tablets of rose hip (ovaltine) being given daily.

The R.B.C. was now 3,900,000 and the haemoglobin 74 per cent.

The left fundus appeared normal and the right fundus nearly so, and there were no fresh haemorrhages.

The patient's general condition was so much better that he was able to come into town for examination, and the following notes were made:—

Right Eye.—V=6/18 and there was difficulty with the letters towards the right side of the line of type, and with −0·50 sph. V=6/18, and there was difficulty with the letters towards the right side of the line of type, and on adding +2·25 sph., near vision was J.6.

The fundus showed a tributary to the superior temporal vein, previously noted as blocked, to be patent now. There seemed to be slight pigmentary changes at the macula, but this was not certain.

Left Eye.—V=6/12 three letters, but not those towards the right side of the line of type, and with $\frac{0·00}{-0·50}$ at 90 deg. 6/12, three letters, but not those at the right side of the line of type, and on adding +2·25 sph., near vision was J.2.

From this it was argued that there was some slight homonymous hemianopia, but it was agreed that his attention should not be directed towards this by carrying out perimetric or Bjerrum's screen examination.

The fundus was normal, except that perhaps the disc edges were a little hazy, but this was not certain.

Summary

A patient with multiple myelomata of the type accompanied by profound anaemia was diagnosed as having subarachnoid haemorrhage, retinal venous thrombosis, and retinal haemorrhages.

Nasal and bladder haemorrhages were also present.

He had received X-ray "spray treatment."

When vitamin P was given the bladder and nasal haemorrhages ceased and there were no fresh retinal haemorrhages, but when the administration of vitamin P was temporarily suspended in September the nasal haemorrhage recommenced, but ceased when administration of vitamin P was recommenced, from which it is argued that vitamin P might be having some therapeutic effect.

By November vision had greatly improved, but there was a homonymous hemianopic depression of the field of vision present. The fundi were practically normal to ophthalmoscopic examination, and showed nothing to account either for the vision still being subnormal or for the hemianopia.

Comments

It is difficult in this case to discriminate justly between reasonable deduction and speculation owing to lack of experience of some of the factors involved. Properly controlled scientific observation is indicated.

It is considered fair to deduce from the evidence available that vitamin P had an effect in this case, influencing the nasal haemorrhage favourably, and presumably the retinal haemorrhage also, but further investigation is needed to confirm this.

The retinal haemorrhages and X-ray treatment.—No fresh retinal haemorrhages occurred after vitamin P was given, one cannot definitely deduce that this was because of the vitamin P, but only speculate.

One can fortify this argument by saying that in retinal venous thrombosis repeated haemorrhages do occur, but that, anyway in this case, in which vitamin P was given, they did not recur. This is still only probability, not proof.

Professor A. J. Ballantyne kindly remarked that the X-ray treatment would increase the capillary permeability, and one can speculate as to whether increased capillary permeability was connected with the subarachnoid and retinal haemorrhages.

X-ray treatment is the only treatment known to influence multiple myelomata favourably, but one of the factors limiting the dosage may be the liability to haemorrhages following increased capillary permeability as a result of the application of the X-rays.

There are no data to show whether, in the present case, there was increased capillary permeability associated with the disease itself, and present before, and independently of, any X-ray treatment, or what the capillary permeability was before and after the X-ray treatment.

But it is suggested that in future cases of multiple myelomata the capillary permeability could be ascertained before X-ray treatment was commenced, and it has been suggested to me that by giving vitamin P before X-ray treatment, and also, of course, during and after it, the capillary permeability could be so controlled as to lessen the danger of haemorrhage after X-rays, and so enable higher X-ray dosage to be used. This could be done in other conditions besides myelomata, in which X-ray treatment is being given.

So far as it has been possible to judge, it would appear that vitamin P has been beneficial therapeutically, and might be beneficial prophylactically, in cases where X-ray treatment is involved.

The question of subarachnoid haemorrhage.—The evidence might seem a little slender. The diagnosis was made before the existence of homonymous hemianopia was known, and was largely on the grounds of sudden headache and retinal haemorrhages, and may be compared with Professor A. J. Ballantyne's cases in the *Brit. Jl. of Ophthal.*, September, 1943.

The hemianopia was discovered late in the course of the illness, and associated with loss of memory suggests cerebral thrombosis or haemorrhages rather than subarachnoid haemorrhage.

There are times when one hesitates to make an out-of-the-way diagnosis and, in this connection, I clearly remember a patient with bilateral thrombosis, when in charge of the Eye Department at the Metropolitan Hospital, London, and of waving a white towel in front of her to see if she could see it. I passed the case on to Dr. C. Worster-Drought, who showed it at the Royal Society of Medicine. (See Bilateral Occipital Thrombosis, Proceedings of Royal Society of Medicine, July, 1928, Section of Neurology.)

In presenting this case an attempt has been made to show it as it grew and developed, rather than to give a summary, though this would have been shorter, of the final findings.

In this connection the fields taken in February, when the patient requested it, are instructive, first of all as showing how far the bedside diagnosis of hemianopia was correct or permanent, and secondly, in connection with the nature of central scotoma in

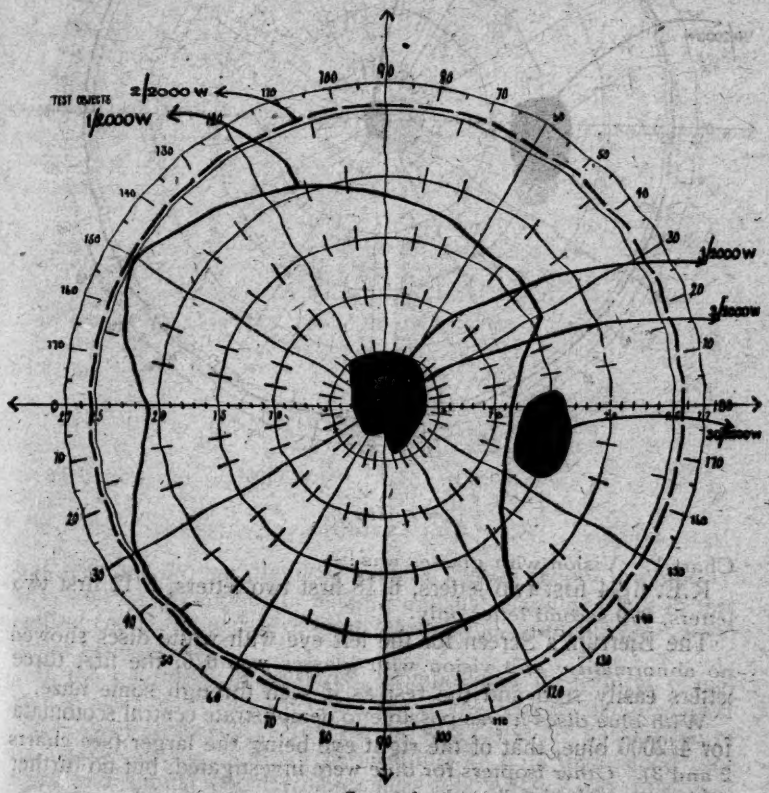

CHART 1.

anaemia, as mentioned in the *Brit. Jl. of Ophthal.*, December, 1943, page 541.

The perimeter fields for white objects were normal.

The Bjerrum's Screen showed for the right eye with white discs baring of the blind spot and a central scotoma with a nucleus.

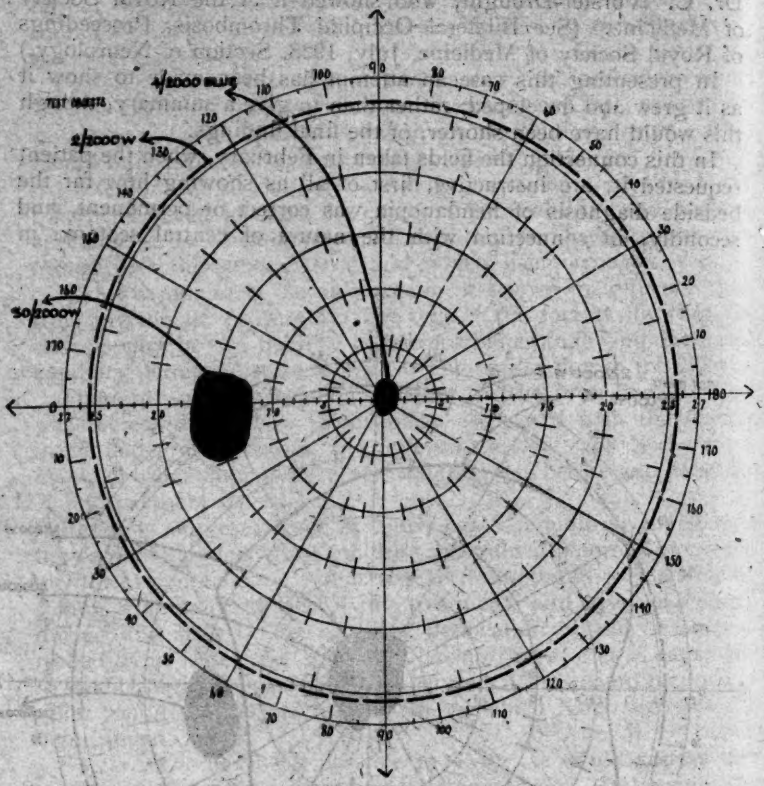

CHART 2.

Chart 1. Vision with glasses was:—

R.E. 6/24 first two letters, 6/18 first two letters, 6/12 first two letters, 6/9 second letter only.

The Bjerrum's Screen for the left eye with white discs showed no abnormality, and vision with glasses was 6/6, the first three letters easily seen and the rest as if seen through some haze.

With blue discs it was possible to demonstrate central scotomata for 4/2000 blue, that of the right eye being the larger (see charts 2 and 3). Other isopters for blue were investigated, but no further information obtained.

Perhaps the papillo-macular bundle has been injured, as mentioned above, in anaemia, but the condition in chart 1 was probably largely associated with retinal haemorrhage.

The cause of the retinal haemorrhages.—(1) *Venous obstruction* of tributaries was certainly present, but by the time the patient

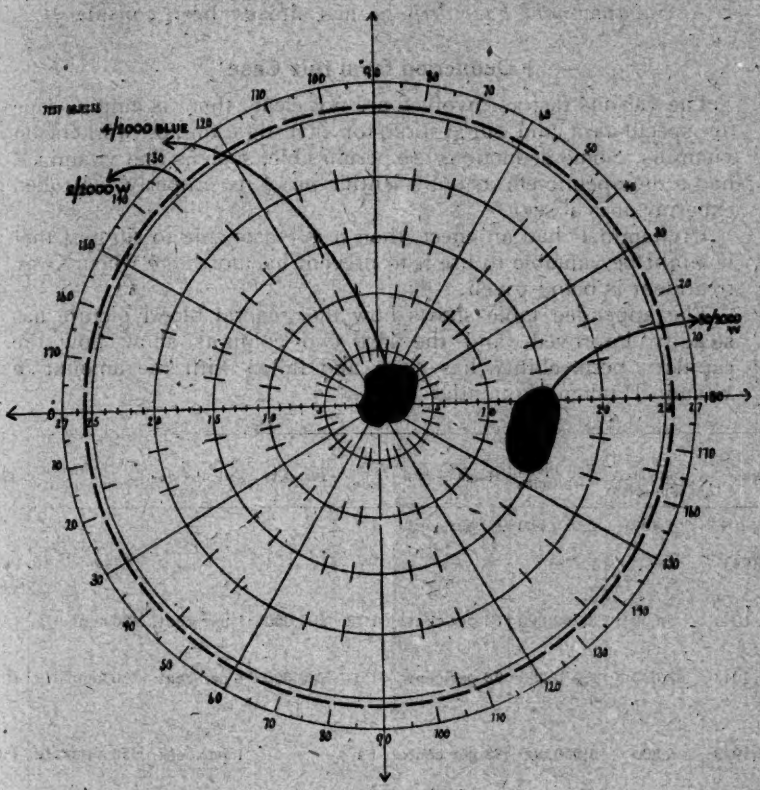

CHART 3.

was seen one did not diagnose bilateral central venous thrombosis. It is well to be cautious in making unusual diagnoses, but in this respect compare the case of bilateral occipital thrombosis discussed under hemianopia.

If the diagnosis of bilateral thrombosis of the central retinal vein is accepted it could account for all the retinal haemorrhages.

(2) *X-ray treatment.*—How far did this cause the haemorrhages by increasing the capillary permeability? It is understood that nasal and similar haemorrhages may follow the X-ray treatment

and that the danger of their occurrence limits the dosage of X-ray that can be used.

(3) *Thrombocytopoenia* has been suggested as causing the haemorrhages as much as the X-ray treatment, and that when the thrombocytes begin to rise, there is a marked and rapid resolution of haemorrhages.

(4) *Subarachnoid haemorrhage* has already been considered.

Deduction from this Case

The various factors involved are not clear; there is ample scope for speculation and suggestion for investigation for workers on vitamins. Some deductions are permissible, such as that vitamin P had a therapeutic effect, but scientific proof by properly controlled experiment is absent.

If vitamin P had an effect, then it is reasonable to suggest that it might be valuable to use it to prevent haemorrhage when X-ray treatment is being given.

The appended table shows how the general blood picture has steadily improved, and the last two columns show how the capillary permeability has risen and fallen with the amount of vitamin P taken by mouth.

Date	White Blood Corpuscles	Red Blood Corpuscles	Haemoglobin	Colour Index	Platelets	Reticulocytes	Capillary Permeability	Treatment
July 26, 1943	4,400	2,030,000	42 per cent.	1·0	—	—	—	—
Aug., 1943	—	—	—	—	—	—	—	Permidin 2 b.i.d. 6/52
Oct. 7, 1943	5,000	2,760,000	64 per cent.	1·18	200,000	1 per cent.	450 mgm. Hg.	—
Oct. 25, 1943	7,600	3,970,000	78 per cent.	1·0	280,000	1 per cent.	250 mgm. Hg.	Rose Hip Tablets 1 b.i.d.
Dec. 15, 1943	6,800	3,950,000	88 per cent.	1·1	—	1 per cent.	350 mgm. Hg.	Permidin 1 b.i.d.

SECOND CASE—General History

The patient first attended hospital in 1936, and from her doctor it was learned that she had albuminuria and myocarditis, but at the time of operation, 1943, she had no albuminuria. She was not robust and appeared to have had the rather hard-worked life which is the lot of many hospital patients. She was of slow mentality, had sallow skin, some facial hair, and a tired appearance on admission, but her mentality brightened and her skin improved in colour after having vitamin P.

Ophthalmological Notes

In 1936 she had early cataract in both eyes, more mature in the left eye and vision with glasses was:—
R.E. 6/12 and L.E. 6/24.
The opacities in the left eye progressed most quickly, till in 1943 it was considered well to extract the cataract.

Operation

On September 14, 1943, a cataract was extracted, with capsulotomy, and a large peripheral iridectomy, but without noteworthy incident, from the eye of a female patient aged 56, and, after needling, vision with glasses was 6/6 (four letters) on November 23.

Post-operative Complications

September 17.—Three days after operation there was a hyphaema filling about a third of the anterior chamber.
September 20.—Iris well dilated and blood absorbing.
September 25.—The blood had absorbed, but haemorrhage had occurred again, there was a considerable amount of blood in the anterior chamber, the aqueous was all pink-coloured, and the iris not visible.
September 28.—Absorption seemed to have commenced, the blood occupying only about 1/5 of the anterior chamber.
The iris was visible, but not well dilated and muddy in appearance, and the whole eye looked unhealthy and as if some catastrophe were impending.
One wondered how often this haemorrhage was going to be repeated and what the prognosis was, for it seemed poor.
September 29.—Permidin (Glaxo), 2 tablets b.i.d. was commenced.
September 30.—The blood was mostly absorbed from the lower part of the anterior chamber, there was less blood in the pupil, the iris was well dilated and bright. The patient herself was much brighter mentally.
October 5.—The anterior chamber was very clear and the clot of blood that had been filling up the pipil was practically absorbed.
October 8.—A spot of pigment about the size of a pinhead remained in the centre of the pupil.
The patient was discharged from hospital and looked much younger and more alert, and her skin had lost its muddy colour and had a more natural and younger appearance.

Comments

There was post-operative haemorrhage, but when after the second haemorrhage permidin was given, not only did the blood

at the foot of the anterior chamber quickly absorb, but a clot filling the pupil also rapidly disappeared, and the general condition of the eye and patient improved.

The clot of blood in the pupil when first seen did not look as if it would be readily absorbed under ordinary circumstances, and it seems possible that the vitamin P given had a beneficial effect on its absorption.

There is no doubt the eye was getting worse till vitamin P was given, but whether it merely commenced to improve at the same time when vitamin P was given, or because vitamin P was given, is not clear.

One thing that is clear is that after vitamin P was given the haemorrhage was not repeated and that the eye cleared up and the general health of the patient improved.

Scientific proof of the value of vitamin P is certainly lacking, but it might be permissible to think that the vitamin P may have been beneficial.

Remarks on both Cases

In both cases there were ocular haemorrhages, and in the first case other haemorrhages also, and the change after giving vitamin P was dramatic. In both cases vitamin P was given, and in both cases not only did the haemorrhages not recur, but the ocular haemorrhages, the ones that could be actually observed, absorbed very satisfactorily. Moreover, in the first case nasal haemorrhage recurred when vitamin P was discontinued at one juncture, and in the second case haemorrhage recurred till vitamin P was given, and ceased thereafter.

It is to be noted that the second case was one of cataract, and that it has been suggested in the American Journal of Ophthalmology, 21/1938, pages 1058 and 1059, that deficiency in vitamin C, with consequent defective lens respiration, may be the real cause of cataract, the whole vitamin C metabolism of the body being deranged in cataract. But the record does not show that any steps were taken to exclude vitamin P, which is apparently often found along with vitamin C in the various natural sources, so that it may be that vitamin P deficiency is a factor in this connection.

In conclusion, it is suggested that there is a case calling for investigation of vitamin P, because, *so far as can be judged* from the evidence of these two cases, vitamin P is of value in ophthalmology.

DESSICATION KERATITIS

BY

Surgeon Lieut.-Commander J. E. M. AYOUB

R.N.V.R.

THIS paper describes a corneal affection which was first noticed amongst the troops of the Eighth Army fighting in the desert during the summer of 1942. The disease is new to ophthalmologists serving in the Middle East Forces, and service conditions have hindered the full investigation and follow-up of cases. It is hoped that this report will lead to general recognition of the existence of the disease so that it may be more fully investigated and prevented.

Incidence.—Most of the cases commenced, according to the histories, during certain periods, after heavy and prolonged fighting had broken out in Libya and the Western Desert in May, 1942.

Judged from the records of this Military Hospital the outstanding dates of onset were (1) the first ten days of June; (2) mid-June; (3) the first week of July. After this there was a gap until October, when two cases occurred.

During the first two summers of the war in the desert no cases were seen. Had any cases been evacuated from the desert in 1940 they would probably have passed through this hospital. In 1941, with greater organisation of advanced ophthalmic centres, they might not have reached here. As methods of examination have not changed, it is unlikely that cases have passed unrecognised in this clinic before 1942. On the other hand, during and after the retreat on the Alamein positions this hospital was close to the line and on an easy line of evacuation for sick and wounded, so that many more patients attended, some as out-patients.

Twenty-five cases have been recorded at this hospital, eight in officers and seventeen in other ranks. One patient only was a naval officer, and it is uncertain whether he was typical. The others were collected from all branches of the Army, and included one South African negro driver. All Army cases commenced in the Western Desert or Libya, with three exceptions, *i.e.*, one started in Syria, one in the canal area, and one officer stated that he had habitually suffered in the same way on manoeuvres in India before the war, though the condition had been unrecognised and diagnosed as astigmatism.

A description of only twenty-five cases probably leads to underestimation of the prevalence of the disease. Captain Martin Williamson, M.C., R.A.M.C., stated in a private communication

* Received for publication, November 22, 1943.

that there was an epidemic of eye complaints which reached serious proportions in the first half of July and during which as much as 10 per cent. of men were suffering. Despite the fact that he had no facilities for examination, he gives such an accurate account of the symptoms and macroscopic signs that there is no doubt that the condition was widespread. He also noted that it occurred especially in observation post officers, drivers, and in patrols—all key men. Therefore the condition is of greater practical importance than this small series suggests.

Symptoms.—These are characteristic and constant. Most intelligent patients described an abrupt onset and could describe the symptoms accurately.

These are (1) smarting and itching of one or both eyes; (2) profuse lacrymation; (3) marked photophobia; and (4) definite disturbance of vision, less frequently described but noted among the more intelligent. This is either a uniocular diplopia, in which a fainter image is seen either above or below an object, such as a distant aeroplane or tank, or else such dark objects have a tail-like prolongation like a comet, or they are vaguely blurred. Where both eyes notice the defect both are found to be affected. This symptom is very definite in some cases where previously eyesight has been excellent, and in several cases the patient has just called with a view to getting spectacles.

Sometimes the symptoms are intermittent, lasting acutely for about three days, clearing up almost completely and then reappearing again. An important point is that the onset, when noted accurately, has been during the day.

Many of the cases, especially those commencing during the fighting around Tobruk in June, were associated with a very marked state of physical and nervous exhaustion.

Objective findings.—These again are very constant. Examination was hindered by lack of a corneal microscope, so that exact localisation of the affected layer of the cornea was uncertain, but from the appearance and translucency of the lesion, and the healing without scarring, it was deduced that only the superficial epithelial layers were affected. This observation has been confirmed by the use of a slit-lamp in the examination of a recent case.

In the active cases there was a marked reddening of the exposed areas of the scleral conjunctiva and a moderate ciliary flush and irritability of the eyes. Although an episcleral injection is a commonplace amongst the desert troops, in these cases it seemed more marked than usual, and it diminished greatly as the cornea healed under treatment.

The corneal changes were of three main types:—

(1) There was a bandlike roughening of the corneal epithelium across the area which would be exposed when the eyes are partially

screwed up as is the case in a bright light. The band was greyish, but translucent and stained with fluorescein, revealing the loss of epithelium. The appearance was punctate. This was the commonest lesion found (Fig. 1).

(2) The lesion was sometimes limited to a few lines of greyish dots, mainly horizontal and in the exposed regions of the cornea. These dots also stained with fluorescein (Fig. 2).

(3) A single stippled line, actively staining, was also seen at times (Fig.3).

In two cases, where the history was very suggestive of this condition and the irregular astigmatism was noted, corneal lesions of the type of Fig. 4 were seen. There was no opacity in the central corneal area, but superficial stippling was seen at the limbal part of the "exposure area." As these cases responded to treatment just as the typical ones did, it is probably correct to regard them as a variety of the condition.

Commencement of healing was shown by the failure of fluorescein to stain the lesions. Some cases of the type of Fig. 1 which were treated throughout were seen to pass through stages corresponding to Figs. 2 and 3 in turn. It is uncertain, therefore, whether Figs. 2 and 3 represent later stages of the condition which starts as in Fig. 1, or whether the condition may commence with the smaller areas of corneal damage. Only examination of cases at the commencement of symptoms will elucidate this point.

On retinoscopy, patients having markedly defective vision were found to have a gross distortion of the retinal reflex. Sometimes the distortion could be seen to be directly due to the band-like lesions; in other cases, especially the Fig. 4 type, the cornea appeared to be clear, but the reflex was wildly irregular, or of "scissors" type. Not only was the marked reflex irregularity present, but it was impossible to correct the affected eyes with lenses subjectively.

The visual acuity improved *pari passu* with the healing. In the active state acuity was often reduced to 6/18 or 6/12. It returned to 6/6 or 6/5 unaided where this had been the previous visual standard, as the condition recovered.

The other parts of the eyes appeared unaffected, and no changes have been noted in lens or retina in any case.

Treatment.—Most cases, especially uniocular ones, have been treated with bandaging of the affected eye to give maximal rest to the cornea. Others where the condition was bilateral have probably done as well with the instillation of castor or cod-liver oil.

Full mydriasis relieves the discomfort rapidly and may hasten healing. Owing to service conditions it has only been possible to follow-up relatively few cases, but they have done uniformly

FIG. 1.

Band-like epithelial loss, staining with fluorescein, a common first stage.

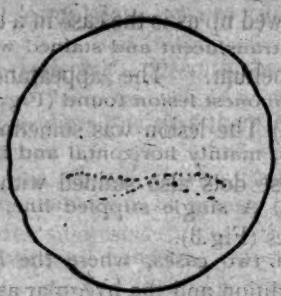

FIG. 2.

Linear punctate epithelial damage. Staining with fluorescein. Sometimes seen as a first stage and sometimes a healing stage

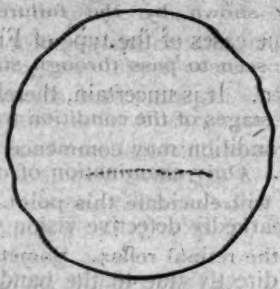

FIG. 3.

A single stippled line. Not always staining with fluorescein. Seen sometimes on first admission to hospital, Sometimes a healing stage.

FIG. 4.

A less common corneal change. Not seen staining actively.

FIG. 5.

Distortion of the retinal reflex as seen with Hamblin's streak retinoscope. Condition corresponding to types 1—3.

CHART TO SHOW CLINICAL DETAILS OF THE CASES QUOTED

Name	Unit	Date Attending	Date of Onset	Place of Onset	Occupation and Conditions at Onset	Side of Eye	Visual Disturbance	Presenting Symptoms: Pain	Presenting Symptoms: Watering	Visual Acuity	Irreg. Astigmatism	Type of Lesion	Treatment	Discharged (D) or Evacuated (E)	Visual Acuity on Discharge
Major B.	R.A.C.	25/6/42	Early June	Lybia, West. Desert	Jeep-riding + + +; period of unconsciousness at onset; ? due to exhaustion; ? due to heat	R	+ + +	+	—	6/18 } not improvable	+	I	Atropine and bandaging at first, later dark glasses	D July 1, 1942	6/5 c̄ +1·25 D.sph. / +0·5 D.cyl. a.v. } still under mydriatic
						L	+ + +	+	—	6/18 } not improvable	+	I			6/5 c̄ +1·0 D. sph. / +0·5 D.cyl. a.v. } still under mydriatic
Capt. D.	R.A.C.	25/6/42	10/6/42	Lybia, West. Desert	Driving with motor transport throughout; very tired	R	+ +	+ +	+ + +	6/12	+	IV	Atropine and bandaging at first, later dark glasses	D June 29, 1942	6/5 c̄ +1·25 D.sph. } still under mydriatic
						L	+ +	+ +	+ + +	6/12	+	IV			6/5 c̄ +1·0 D.cyl. } still under mydriatic
Gnr. G.	N.Z.A.	5/7/42	Early June	Syria	Noticed after ride on roof of railway cab; hot day	R	—	—	—	6/5c̄ – 1·25 D.cyl. 15°	—	—		E	
						L	—	+ +	—	6/5c̄ – 0·75 cyl.	-	II			
L./Bdr. B.	S.A.A.	7/7/42	1/7/42	West. Desert	—	R	+ + +	+ + +	—	6/24		I		E	
						L	+ + +	+ + +	—	6/24		I			
Rfm. R.	B.I.	7/7/42	1/7/42	West. Desert	—	R	—	+ +	+ +	?		II		E	
						L	—	+	+	?		—			
Lt. C. ...	R.A.S.C.	7/7/42	7/6/42	Knightsbridge, West. Desert	Continuous driving; very fatigued on admission	R	+ + +	+ +	+ +	6/36	+	I	Atropine, dark glasses	D July 11, 1942	6/6 (c̄ – 2·5 D.sph.)
						L	+ + +	+ +	+ +	6/24	+	II			6/6 (c̄ – 1·75 D.sph. / +1·0 D.cyl. a.v.)
Pte. C. ...	B.I.	8/7/42	Early June	Gazala, West. Desert	—	R	+ +	—	—	6/5	-	III		E	
						L	+ +	—	—	6/60 amblyopia exanopsia	-	III			
Dvr. R.	R.A.	8/7/42	Early June	Lybia	Driving a lot during the retreat	R	—	+ +	—	6/6		—	Hyoscine in oil, flap	E	
						L	+ +	+ +	—	6/12 –		I			
Cpl. S. ...	S.A.S.C.	9/7/42	?	Gazala, Lybia	Retreat from Gazala: no goggles worn	R	—	+ +	—	6/6 } corrected		III	Hyoscine in oil, dark glasses	E	
						L	—	+ +	—	6/36 } corrected		II			
Cpl. H....	S.A.A.	9/7/42	1/7/42	West. Desert	—	R	+ + +	—	—	6/60 } unchecked		III	Hyoscine in oil, dark glasses	E	
						L	+ + +	—	—	6/36 } unchecked		III			
Dvr. S....	S.A.S.C.	13/7/42	Early or Mid-June	West. Desert	Retreat from Gazala	R	—	+ +	—	?		II-III	Hyoscine in oil, dark glasses	E	
						L	—	+ +	—	?		II-III			
Rfm. N.	B.I.	13/7/42	7/7/42	West. Desert	Over varying terrain—mostly yellow sand	R	+ +	+ + +	+ + +	? under Mydriatic		II	Ol. Morrhuae only flap	E	
						L	+ +	+ + +	+ + +	? already		—			
Gnr. D....	N.Z.A.	23/7/42	21/7/42	West. Desert	Sandstorm on day of onset; drives a truck	R	—	+ +	+ +	6/6		III	Ol. Morrhuae	E	
						L	—	—	—	6/6		—			
Sgt. W....	R.A.	24/7/42	Early June	West. Desert	Much travelling on columns	R	+ +	—	—	6/9 –	None	I	Hyoscine in oil bandaging	E	6/6 c̄ +0·5 D.cyl. a.v.
						L	—	—	—	6/36 (L.C.C.S.)		—			6/24 c̄ +2·0 D.sph.
Gnr. S....	R.A.	24/7/42	7-14/6/42	West. Desert	Driving a long distance; watching the view; clear day; no undue heat or dust; white sand	R	—	—	—		-	—		E	
						L	+	+ + +	+ +	6/9		I	Hyoscine in oil bandaging		
Pte. A....	B.I.	25/7/42	1. Early June 2. relapse, 22/7/42	Gazala Alamein	Out in sun at look-out post; v. hot day; fainted owing to the heat; no sandstorm; brown soil; shells raising some dust around him	R	—	—	+ + +	6/6				E	
						L	+ +	+ +	+ + +	6/18	+	I	Hyoscine in oil bandaging		
Major L.	B.I.	25/7/42	22/7/42	Alamein	Driving + +; white sand; hot dusty day; v. tired; slept on a beach	R	—	+ +	+ + +	6/5 –		III	Hyoscine in oil bandaging	D July 28, 1942	6/5 easily
						L	—	—	—	6/5		—			6/5
Tpr. D....	R.A.C.	27/6/42	13/6/42	Lybia and West. Desert	Driver of truck	R	—	—	—			—		E June 27, 1942	
						L	?	—	+ + +	6/36	+	I	Atropine bandage		
Sgt. D....	R.A.	1/8/42	1/7/42	Lybia and West. Desert	Driving a lot on mobile cols.; noticeably exhausted	R	—	+ + +	+ +	6/9		I	Hyoscine in oil dark glasses	E	
						L	—	+ + +	+ +	6/9		I			
Capt K.	N.Z.M.C.	7/8/42	27/6/42	Alamein	Sudden onset on Fuka aerodrome while standing about; v. hot day; had had little sleep for a week	R	+ + +	+ + → —	+ + → —	6/12c̄ lenses	+	IV	Hyoscine in oil, dark glasses	D Aug. 17, 1942	6/5 c̄ lenses, 6/6 unaided
						L	+ + +	+ + → —	+ + → —	6/18c̄ lenses	+	IV			6/5 c̄ lenses, 6/6 unaided
Tpr. L....	R.A.C.	10/8/42	Early June	Knightsbridge, West. Desert	Fighting and driving for severe spells	R	+ + +	+ +	+ +	6/24	+	I-II	Hyoscine in oil, dark glasses	D Aug. 13, 1942	6/6 unaided
						L	—	—	—	6/6	—	—			6/6 unaided
Tpr. H.	R.A.C.	11/8/42	Early July	Alamein	At onset was not travelling; yellow sand; no sandstorm	R	+ + +	+ + +	+	6/18 –	+	IV	Hyoscine in oil, dark glasses	D Aug. 13, 1942	6/6 c̄ +0·75 D.sph.
						L	+	—	—	6/18 +	+	IV			6/6 c̄ +1·0 D.sph.
Major E.	R.A.C.	14/11/42	Oct. 1942	West. Desert	Previous recurrent attacks while riding on manoeuvres in India; started again this time while on intensive training; driving all the time	R	+ + +	—	—	6/12	+	IV	Hyoscine bandage	D Nov. 22, 1942	6/5 c̄ +0·75 D.sph. / +0·25 D. cyl. → 110
						L	—	—	—	6/6	-	—			
Lt. S. ...	B.I.	14/11/42	Oct. 1942	Canal Area	Fair amount of training recently, at start of symptoms	R	+ + intermittent	+ + +	—	6/6 –	+	II	Ultimately required carbolising	D Nov. 23, 1942	6/6
						L	—	—	—	6/6		—			
Lt. Cdr. F.	R.N.	30/11/42	Early Nov. 1942	At Sea	First noticed after a gun-blast from that side. At first treated as conjunctivitis, but visual acuity poor throughout	R	+ + +	+ +	+ +	6/24	+	I	Hyoscine bandage	D Nov. 16, 1942	6/5 unaided
						L	—	—	—	6/6					

well. In all except one case the cornea has been healed and clear, and the visual acuity restored to normal in between five and seven days. The patients have been fit for duty, and those of whom news has been received have remained well and active since their return to the desert.

Discussion of Aetiology.—Three points strongly suggest that the lesion was caused by some physical factor :—

(1) The invariable localisation of the lesion to the small area of the cornea exposed by the eyelids when screwed up as protection against the bright sunlight of the desert.

(2) Its resemblance to the results of " flash " burns affecting the eyes and the vesiculation, caused by ultra-violet light emanations acting on the cornea, and to keratitis e lagophthalmo.

(3) The fact that the majority of the patients gave a history of driving in open vehicles just before or at the start of the symptoms.

The case which arose in Syria was in a soldier who travelled on a very long train journey on a hot day, sitting outside the railway carriage.

In another patient, an artilleryman, the condition started at the end of a drive from Mersa Matruh to Halfaya Pass, in which he had been admiring the scenery approaching the famous pass by looking over the top of the driver's cab.

A minority of the patients did not give a history of driving, but had been in a very exposed position in the desert and had noticed the heat unduly.

In one case, the naval officer, the onset of his visual disturbance and inflammation one month previously, which was diagnosed as conjunctivitis at first, was just after receiving the blast of a gun on the same side as the affected eye. He was found to have the corneal change of Fig. 1 type and irregular astigmatism of a typical case, and therefore he is included in the list, although he was not exposed to the desert conditions.

Discussions of the cause of the lesions.—The literature available shows that a similar condition has been described in cases of snow-blindness. Würdemann (" Injuries of the Eyes ") mentions the presence of a corneal erosion in this condition. G. L. Struder (Ophthal. Rec., November, 1906) recorded six cases of snow-blindness occurring during a blizzard in North America, where a corneal erosion could only be found by staining with fluorescein. He mentions that Graddy, of Nashville, Tenn., had previously described a similar condition due to exposure on snow fields. These observers ascribed the condition to ultra-violet emanations reflected from the snow.

In this connexion, a very good description of the condition has been given by Halphen (and Sedan), who noted a practically

horizontal opacity "keratite en bandelettes" and stainable by fluorescein. The opacity remained for ten days. They say "one might call the superficial alterations 'steamy cornea'—buée epitheliale."

Duke-Elder, in describing photophthalmia ("Rec. Adv. in Ophth."), writes that it may be caused by sunlight when reinforced by surfaces such as sea, desert, or snowfield.

In the investigation of photophthalmia produced experimentally the first experiments were made by Widmark (1889-1903), using an arc-light, which produced a reaction in the epithelial cells and to a lesser degree in the substantia propria of the cornea. With heavy doses lenticular opacities were produced. Birch-Hirschfeld, Kertel, Breose, Ognell, and Strebel found only corneal but no lenticular changes. Verhoeff and Bell, Hess and Duke-Elder have, however, produced lenticular changes.

In view of the changes found in experimental and clinical photophthalmia it has been assumed that desert and snow-blindness are due to the action of ultra-violet light reflected into the eyes.

In the literature at my disposal no description has been found of any similar condition occurring in a desert, and no mention has been made of the effect of hot air or wind playing on the exposed surface of the eye.

Possible Causative Factors.—The following may be considered:

(1) Damage by light radiations of short wave-length, *i.e.*, photophthalmia.

(2) Damage of the epithelium by driven sand, either in a sand-storm or raised by vehicles driving in front or by shelling.

(3) The action of irritant gases.

(4) Drying of the exposed area of the cornea by intensely hot air or wind.

(5) Undue exposure of the affected area of the cornea by fatigue, which reduces the frequency of the blinking reflex of the lids. This would predispose to factor 4.

Vitamin deficiency may be ruled out, as these patients have all been in good bodily condition and showing no sign of other defects, apart from physical exhaustion in several cases.

(1) *Photophthalmia.*—Three points are strongly against this being the cause. First, the outstanding feature of clinical photophthalmia is the well-marked latent period between exposure and development of symptoms, corroborated by Duke-Elder's experimental irradiation of the corneae of rabbits. In quite a large proportion of the cases under review, and especially noticeable in those men who gave an intelligent history, the onset of the symptoms was *during* the period of exposure.

Secondly, since the soil of different parts of the desert varies

considerably in colour, one would have expected the whiter sands of the El Alamein area to reflect more ultra-violet light than the browner soil of the Tobruk area. Actually more cases occurred around Tobruk than in any other single area.

Thirdly, no cases have been noted in the Mediterranean Fleet, where the conditions of service undoubtedly expose the eyes of the sailors to a great deal of radiation.

It is improbable, therefore, that this condition is a type of photophthalmia.

(2) *Damage by sand.*—Although dust was blamed by many victims, in at least three cases it could not have been a factor. These were the men who developed it on the train in Syria, the gunner who was admiring the view of Halfaya, and the naval patient who received the blast of the gun. Other cases have also stated that they did not consider it unduly dusty when the condition started. Where patients have ascribed the onset to a sand-storm they have also admitted that it was a day of particular heat (a typical weather combination in Egypt). Indeed, one patient did have treatment for heat-stroke on the day that the symptoms started, and another also stated that he had " passed out " for some hours from an unknown cause, either the heat or exhaustion, on the date of the onset.

It is impossible to find enough cases to suggest that sand is not an invariable factor.

It is noteworthy that in no case was any sand particle found embedded in the cornea, nor were any ulcers seen suggestive of this having occurred, and, again, foreign particles in the conjunctival sac do sometimes produce tiny superficial abrasions where the lids rub them against the corneal epithelium. In these cases the line of each individual scratch appears to be vertical, whereas in the keratitis described the spacing of individual grey dots in the band-like area affected was quite regular and not arranged in vertical linear bars,

The general appearance of the lesion was so similar to those resulting from cocaine, exposure to air and flame, that one could not help feeling that sand was an unlikely cause by comparison.

(3) *Irritant Gases.*—In no case could a history of exposure to fumes of any sort be elicited, apart from the smoke of shells in a few cases.

(4) *Drying of the cornea.*—The appearance of the lesion being that of one with a physical cause leads to the conclusion that drying of the cornea by the hot, dry air of the desert is the probable cause. Three points strongly favour this hypothesis:—

(1) The majority of the patients have been riding open vehicles at the start of the symptoms.

(2) In no case in the records have glasses or goggles been worn at the time of onset.

(3) This is the first occasion on which there has been prolonged and hard fighting in the hottest part of the year, with short nights, permitting little sleep.

(5) *Undue exposure of the cornea from fatigue.*—It was most noticeable that these patients seen in the early stages were extremely fatigued, and looked mere shadows of the men they became after three days in hospital. The officers and N.C.O.s, who were of an energetic and keen type, admitted that before the start of the symptoms "things had been pretty hectic," to use a typical understatement. It is also noteworthy that, with one or two exceptions, all the men had come from forward areas, the majority being from fighting units who had been in the thick of the fray.

It is possible that in cases of great fatigue blinking may be reduced in frequency. This reflex is a most important factor in moistening the cornea, and if it were reduced would pre-dispose to drying.

Discussion

From a consideration of the above points it is probable that the physical cause of the lesion is drying of the corneal epithelium. The weather conditions in North Africa are such that the average temperature rises as the summer progresses, but in the early part of the summer spells of southerly winds with high temperature and low humidity are frequent. During these spells the wind has a great drying effect on the skin and feels noticeably hot as it blows on the eyeballs.

It is possible that similar lesions, ascribed to photophthalmia when occurring on snowfields, are again at least in part due to desiccation and perhaps to freezing as well. One or two men who claimed to have suffered from "snow-blindness" in the Archangel expedition of 1919 have been interrogated, and stated that the onset was during long drives and that there was no latent period between exposure and the onset of symptoms. This suggests that "snow-blindness" may not, in some cases at least, behave like a true photophthalmia, and that it may have been wrong to ascribe the band-like corneal opacity to the effect of ultra-violet light radiations.

(1) Temporary damage has been caused to the eyes of soldiers serving in the North African desert. The site is the corneal epithelium and the condition is transient.

(2) Discomfort, photophobia and impairment of vision are the outstanding symptoms.

(3) It is probable that the cause is drying of the corneal epithelium by a hot, dry wind, which causes excessive evaporation of the lacrymal secretion over the exposed area of the cornea.

(4) The suggestion is put forward that corneal damage ascribed

to snow-blindness is really caused, certainly in part, by desiccation and perhaps also freezing of the corneal epithelium, and not by excessive ultra-violet light reflections.

(5) The condition could be prevented by the wearing of goggles.

(6) The name desiccation keratitis is suggested for the condition.

I would like to express my gratitude to Brigadier C. W. Graham and Brigadier G. I. Scott and to Dr. Rowland Wilson, of the Giza Memorial Hospital, Cairo, for their great help and advice in the preparation of this paper, and to Colonel J. Morrison, O.B.E., M.C., for permission to publish it.

THE SECRETION-DIFFUSION THEORY OF INTRA-OCULAR FLUID DYNAMICS*

BY

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RECENTLY Duke-Elder and Davson[1] discussed certain phases of our paper entitled " The mechanism of aqueous humour formation inferred from chemical studies on blood-aqueous humour dynamics "[2]. From their comments it is apparent that these authors obtained the wrong impression concerning our viewpoint. Perhaps as a consequence they incorrectly modified certain equations which we had set up to represent the ultra-filtration mechanism of aqueous humour formation. We feel therefore that some recapitulation of our theory of intra-ocular fluid dynamics is in order. At the same time we wish to discuss the criticisms which Duke-Elder and Davson have made concerning the mathematical concepts we have used in developing our theory.

Through the use of heavy water (D_2O) as a tracer substance for ordinary water it was shown that one half of the water in the anterior chamber of the rabbit was replaced in slightly less than three minutes[3]. This is equal to a total water movement into and out of the anterior chamber of approximately fifty cubic millimetres per minute, a rate, it will be observed, far in excess of that which was thought to correspond to the rate of formation of aqueous humour as a whole. This finding suggested that the various constituents of the aqueous humour may enter the anterior chamber at different rates, and that it was unrealistic therefore, to speak of a rate of formation of aqueous humour as a whole. To

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test this hypothesis the rates of accumulation of sodium and of chloride in the anterior chamber were measured under strictly physiologic conditions by employing radio active isotopes of these elements.[4] From the rate of accumulation of these ions it appeared that their rate of entry from the blood was equivalent to approximately four cubic millimetres of whole aqueous humour per minute. From the difference in rate of movement of water and these two ions it appeared certain that in the formation of aqueous humour all the constituents do not enter at a common rate, *i.e.*, "as a whole," but that they are transferred from blood to aqueous humour at rates which are characteristic for each substance.

In order to test this idea further, the rates of accumulation in the aqueous humour of various other substances were determined[5]; these included the ions thiocyanate, bromide, lithium and phosphate, and the non-electrolytes, urea and levulose. A record of the concentrations of these substances in the plasma was obtained and the concentration in the aqueous humour was determined and related to the maximum diffusible concentration in the plasma of each animal.

Having obtained these experimental data we then set out[2] to see whether they were consistent with what would be expected on the basis of the following postulated mechanisms. First possibility.: constituents of aqueous humour separately may enter the anterior chamber from the blood by a process of ultra-filtration (diffusion) where, under static conditions, they would accumulate until the concentration in the aqueous reached that in the blood. Second possibility: they enter by a process of ultra-filtration but leave by another mechanism, *e.g.*, flow. *Third possibility: they* enter by a secretory process, in which instance it is necessary that there be some leakage (flow) out of the anterior chamber. It was pointed out that some combination of these processes (or others) might actually be involved in aqueous humour formation.

Equations consistent with the above possible mechanisms were formulated relating rates of transfer of a substance into and out of the anterior chamber to the ratio of its concentration in the aqueous to that in the blood at various intervals after its introduction into the blood.

Then for each substance tested a determination was made of the best fit obtainable on the basis of each of the above assumptions for every experimentally determined concentration in the aqueous humour. This fit was determined by plotting the rate of transfer into the aqueous humour against a series of assumed rates of transfer out of this chamber for all of the experimentally found concentration ratios on the basis of both the ultra-filtration and secretory mechanisms. This method of treatment gave rise to two sets of values, one set for each mechanism.

It was found that all the experimental data agreed well with an assumed rate of leakage out of the anterior chamber of approximately four cubic millimetres per minute. Thus, of the total water movement (fifty cubic millimetres per minute) between the blood and aqueous humour, only about one-twelfth could be accounted for on the basis of a through and through circulation; the remainder appears to be simply exchanged between blood and aqueous. It was found, furthermore, that the data resulting from tests made with the electrolytes were compatible only with the idea that all of these substances entered the anterior chamber as a result of a secretory process. While it could not be decided on the basis of these experiments whether the non-electrolytes enter the anterior chamber by ultra-filtration or secretion, it is our opinion, based to a considerable extent on the experimental studies of others, that non-electrolytes enter the anterior chamber by ultra-filtration. Because of our lack of exact knowledge concerning the pressure relationships it would appear better to substitute the more general term diffusion for the term ultra-filtration.

Thus we can summarize our present concept of aqueous humour dynamics by stating our belief that the *electrolytes enter* the anterior chamber as a result of *secretion; the non-electrolytes enter* by *diffusion*. Both *electrolytes and non-electrolytes*, we believe, *leave* the anterior chamber by a *process of flow*.

The process may be pictured as follows: the electrolytes are transferred as a result of a secretory process from the blood to the aqueous humour across a barrier which appears to be essentially impermeable to the simple diffusion of ions. The nature of the mechanism by which such secretion may take place has been discussed by Friedenwald.[6] The electrolytes are secreted at a rate which is apparently sufficient to maintain their concentration in the aqueous humour slightly in excess of that in the blood—so that the total osmotic activity is equivalent to about 5·4 m.M. of sodium chloride per kilogram of water higher than in the blood[7]. This hypertonicity accounts for the continual passage of water into the eye, and also, we believe, for the intra-ocular pressure. The magnitude of the pressure is normally limited by the continuous loss of fluid by outflow from the anterior chamber.*

The non-electrolytes, like urea and glucose, enter the eye in a manner similar to water, *i.e.*, by diffusion, and do so at a relatively slower rate than water. Accordingly, since there is an outflow of aqueous humour the concentration of non-electrolytes is normally less than in the blood. This may be seen more readily perhaps by considering the following hypothetical case.

* Were there no outflow, the difference in osmotic activity is sufficient to produce a pressure in an otherwise normal eye of about 165 millimetres of mercury. Any increase in the permeability of the blood-aqueous barrier results in lowered intra-ocular pressure due to abolition of the osmotic pressure difference.

Accepting the rate of leakage out of the anterior chamber of four cubic millimetres per minute, let us consider a substance (non-electrolyte) which exists in the blood in a concentration of 0·01 milligram per cubic millimetre, *i.e.*, one part per hundred. Suppose that this substance has such properties with respect to the blood-aqueous barrier that it enters the anterior chamber at a rate of 0·005 milligram per minute, that is to say, one part per eight hundred of water. (It is, of course, because of the hypertonicity of the aqueous humour that it is possible for water to enter the eye disproportionately faster than the non-electrolytes). It may readily be calculated from equation (1 a) or (4) which appear in a following section that under these conditions the concentration of the substance in the aqueous humour will be only 11 per cent. of that in the blood. Were the rate of entrance of the substance still slower relative to water, as would be the case for a larger molecule, or if the rate of flow out of the anterior chamber were more rapid, the concentration of the hypothetical substance in the anterior chamber would be still less.

Duke-Elder and Davson, in presenting their criticism, apparently failed to distinguish our consideration of the various possible mechanisms from our subsequent conclusions as to which, if any, of these mechanisms were in agreement with our experimental data. They limited their discussion to one of the mechanisms which we had considered, the one consisting only of ultra-filtration and outflow, and claimed that such a simple mechanism is improbable because of the energy requirements. Apparently they did not appreciate that such a mechanism was considered only in order to determine how it compared with the other mechanisms in accounting for the observed behaviour of individual test substances. They cite the mathematical development which we present to make this determination*, and state (page 433) that we are in error in our argument because we integrate equation (1 a) at a constant volume. On the contrary, the integration of equation (1 a) at a constant volume is valid since there is no question of any change in the volume in the eye. Duke-Elder and Davson introduce a replacement concept which is

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* (1) $\frac{dA}{dt} = k_2(C_1 - C_2)$ — C_1 equals concentration in the blood.
†C_2 equal concentration in the aqueous humour at any time.

(1*a*) $\frac{dA}{dt} = k_2(C_1 - C_2) - k'C_2$ (where A equals the amount of constituent present in aqueous humour).
when $t = \infty$

(4) $\frac{C_2}{C_1} = \frac{k_2}{k_2 - k'}$

† Duke-Elder and Davson properly call attention to our original somewhat confusing notation. C_2 should equal concentration in aqueous humour at any time instead of at a given time, t_1.

based on the argument that the condition of constant volume requires the addition of "fluid" (containing the substance in question) in amounts over and above that which is consistent with equation (1), *i.e.*, than may be accounted for on the basis of the ultra-filtration process. It is our contention that such an argument is fallacious. Their equation (5)* which, like our equations, deals only with a constituent and not with fluid, states that an additional amount of this substance will enter the anterior chamber by a process different from that which we had postulated, viz., one of ultra-filtration, wherein by definition, the rate of change of A is entirely dependent upon the difference in concentration of a constituent between blood and aqueous humour. They believe the constituent will enter in addition by a process described by the factor $k'C_1$. It is clear that such a hypothetical process is completely different from one of ultra-filtration which was originally postulated.

Now with regard to the replacement of fluid lost from the anterior chamber, we already discussed this problem in connection with these particular equations and said "moreover, it is assumed that the relative ease with which water may move across the barrier in either direction, as shown in[3], permits the volume, hence the intra-ocular pressure, to remain essentially constant despite loss of fluid by means of flow."

That Duke-Elder and Davson's whole argument is invalid can perhaps be more readily visualized through the use of a simple model. Suppose we picture a tank (with infinite capacity to be replenished) filled with water containing suspended particles. To one side of this tank is joined a second smaller tank which is separated from the first by a screened partition which will permit the slow exchange of these particles and the rapid exchange of water between the two tanks. Attached to the smaller tank is a tap so that water and particles may be drained off at will. The particles will eventually distribute themselves evenly throughout the two tanks provided there are no leaks. If, however, the tap is opened and left open (analogous to flow out of the eye) the concentration of the particles in the second tank will decrease and when a steady state is reached the relative concentrations in the two tanks will clearly depend on the relative rate of passage of particles from the larger to the smaller and out of the latter.

Applying Duke-Elder and Davson's comments to our model they would say that if the volume in the smaller tank is to remain constant the fluid lost from this tank must be replaced by fluid from the first tank, and indeed this is true, but it will *not* "clearly increase the rate of increase of A, the amount of substance in the

* $\frac{dA}{dt} = k_2(C_1 - C_2) - k C_2 + k'C_1$.

aqueous" (second tank) "by a factor $k'C_1$". Moreover, it is *not* necessary to argue, as they suggest, that the fluid which replaces the aqueous humour lost *via* Schlemm's canal is *free* from the particular (dissolved) substance. Referring again to our model the rate of entrance of the particles (assuming a diffusion process analogous to that in the eye) will be proportional to the difference in concentration in the two tanks just as we postulate in equation (1 a). The effect of an increase in the rate of outflow will be to lower the relative concentration in tank number two. This will increase the difference between C_1 and C_2, but it will not mean that the physics of the whole model will be changed so that, in addition to particles entering due to the assumed diffusion process, they would also enter at a rate proportional to the concentration in the first tank, as Duke-Elder and Davson stated would occur in the eye.

In the last paragraph of their paper Duke-Elder and Davson claim to restate their criticism for the benefit of the non-mathematical reader. They say that "if the concentration of a given substance, say urea, is higher in the blood than in the aqueous humour, the system plasma-aqueous humour, is not in equilibrium and hence diffusion of urea should take place into the aqueous humour until the equilibrium condition of equal concentrations in the two fluids is achieved." We agree with this statement provided there is no drainage. They then say that we claim that "as soon as drainage occurs the concentrations become different." Perhaps they would have expressed our view better had they said that "if we assume that there is drainage the concentrations never are equal." However, with the general idea we appear to be in agreement. Duke-Elder and Davson then proceed with their argument by saying that "the second law of thermodynamics states that a system of this kind can only be maintained at a non-equilibrium position by the continued performance of work on it. Drainage of fluid away from the anterior chamber is not capable of performing the necessary work on the system" they say, "so that in the absence of secretory activity the concentration differences claimed by Kinsey and Grant are excluded by the second law." We agree with their statement of the demands of the second law of thermodynamics, and also with their idea that the concentration difference which we found for non-electrolytes would be excluded by this law were there no secretory activity. Since we have repeatedly expressed the conclusion in our paper[2] that "all the monovalent electrolytes tested entered the anterior chamber as a result of a *secretory process*," it would appear that the argument, if indeed it be one, against our theory of intra-ocular fluid dynamics is not relevant.

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THE LATE RESULTS OF REMOVAL OF INTRA-OCULAR FOREIGN BODIES WITH THE MAGNET*

BY

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THERE have been few recent publications of the late results of the removal of magnetic intra-ocular foreign bodies, so that a follow-up of the cases treated at Moorfields during the first 4 years of the war may be of general interest.

Of the 154 cases in which a magnetic foreign body was successfully removed from the eye during that period, the posterior route was used only twice, and of these, one eye became shrunken and siderotic, and the other required enucleation early from sepsis. Removal through the scleral wound of entry was effected in 2 further cases, the resultant vision being reduced to 6/18 from a localised cataract and 6/60 from macular striae respectively.

In the remaining 150 cases, the anterior route was employed. In about 15 per cent. of these the foreign body lay in the anterior chamber, and in the remainder it was drawn thither by the Haab magnet (or in 3 cases only by the Mellinger), final removal being effected with the hand magnet through a keratome section. The vision was estimated when the eye condition could be considered static 6 months to 3 years later, except in the very few cases in which the patient was out of reach, when an earlier record had to be accepted.

* Received for publication, February 23, 1944.

RESULTANT VISION

	No. of cases	6/5–6/6	6/9	6/12	6/18	6/24–6/60	C.F.	H.M.	P.L.	No P.L.	Excision
Anterior route extractions											
1. With lens undamaged	48	17	14	3	3	4	3	2	2	0	0
2. With lens damaged:											
(*a*) cataract not removed	85	5	2	2	5	6	12	15	9	2	27
(*b*) cataract evacuated later...	17	2	5	2	2	3	1	1	1	0	0
Total cases	150	24	21	7	10	13	16	18	12	2	27

This may be summarised as follows:—

RESULTANT VISION

	Good (6/5–6/9) per cent.	Moderate (6/12–6/60) per cent.	Bad (<6/60) per cent.	Excision per cent.
1. With lens undamaged	65	20	15	0
2. With lens damaged	13	20	40	27
Of total by anterior route	30	20	32	18

Factors Influencing the Resultant Vision

(1) *Damage to the lens* gravely affected the prognosis as is seen in the foregoing table, two-thirds of those with lens undamaged attaining good vision (6/9 or better), and two-thirds with lens damaged attaining bad vision only (less than 6/60).

(2) *The amount of uveal damage* was of considerable importance in determining the amount of permanent visual loss; in the following table those cases with holes and tears of the iris, traumatic hyphaema, iris or ciliary prolapse, hypopyon iritis or gross synechiae are arrayed against those with uveae intact and their resultant visions recorded:—

RESULTANT VISION

	Good (6/5–6/9)	Moderate (6/12–6/60)	Bad or Excision (<6/60)	Total
Without uveal damage	30	18	21	69
With uveal damage	8	11	49	68

(3) *The site of the entrance wound* has also some influence, those with the wound at the limbus having the worse prognosis, and those with scleral wounds (especially when within 5 mms. of the limbus) less favourable than those with corneal wounds.

RESULTANT VISION

	Good (6/5 – 6/9)	Moderate (6/12 – 6/60)	Bad or Excision (<6/60)	Total
Wound of entry in cornea	32	22	46	100
Wound of entry across limbus ...	2	1	8	11
Wound of entry in sclera	5	2	8	15

(4) *Delay in removal of intra-ocular foreign body* did not seem materially to prejudice the issue, although in two cases siderosis was evident which could have been averted. This is made clear by the following chart, in which the proportion of cases that attained good or moderate vision to those with bad vision remains constant irrespective of delay. Excisions were, in fact, relatively more frequent in those cases reaching hospital early; but this may be explained by the fact that the more severe injuries with the worse prognosis would demand hospital attention more urgently.

	Number obtaining	Good or moderate vision 6/5 – 6/60	Bad vision <6/60	Excision
Delay in attending hospital, 0 days		25	17	18
Delay in attending hospital, over 1 day		20	13	5
Delay in attending hospital, over 4 days		15	7	5
Delay in attending hospital, over 14 days		11	4	1
Delay in attending hospital, over 2 months		5	3	1

Commentary

Lens damage was produced in two-thirds of the cases, although in 14 of the 102 the cataract remained localised enough to allow reading sight (>6/18). Of the remaining 88, excision of the eye was necessary in 27, representing 18 per cent. of the total cases, usually within a few days owing to suppuration, but in a minority, after a few weeks owing to the risk of sympathetic ophthalmia.

In 17 cases the cataract was subsequently removed, and ten of these failed to regain good vision owing to iritis of varying intensity.

Of the 48 cases in which no lens damage was caused, in 16 the vision was impaired, the following factors being responsible:—

Vitreous haze or haemorrhage	6
Retinal commotio	5
Retinal detachment	1
Pigment on antr. lens capsule	1
Ciliary prolapse with subsequent glaucoma	1
Corneal scar	1
Previous amblyopia (F.B. present 35 years although no evident siderosis)	1

In comparing these figures with others previously published it is noted that Anderson in 1936 found excision required in 33 per cent. of anterior route extractions and 50 per cent. of posterior route extractions and in the remainder the various degrees of impairment that he found were in proportions corresponding to those recorded above.

He also observed that although the large majority reached hospital within a day of the accident a delay did not appear to have much ill effect on the ultimate vision; this is confirmed by the cases treated at Moorfields, where of the 150 cases in which the time lag was recorded, 60 reached hospital the same day and 28 the following day.

A survey of the magnetic intra-ocular foreign-body extractions performed at Moorfields was published in the Moorfields Hospital Reports of 1897 by Mr. Mackenzie, and again in 1907 by Mr. Goulden, and provide an interesting comparison with the present day figures. During the 6 years (1890 to 1896) 50 foreign bodies were removed; in 10 cases the foreign body did not penetrate beyond the iris and good vision resulted; of the remaining 40, 20 lost the eye, and among the remainder on their discharge, a third allowed good, a third moderate, and a third bad vision (using the standards previously mentioned).

The Mellinger magnet has fallen largely into disuse at Moorfields, as it is usually considered more dangerous and cumbrous to apply, and in addition considerably weaker than the Haab.

It has been argued that by using the anterior route there is a risk of damaging a previously undamaged lens; in 4 cases only of the 150 has subsequent lens opacity been noted in a case in which the lens was apparently clear on admission, in two of these the cataract remained small and peripheral and in a third siderotic lens changes ensued; having regard to the frequent difficulty in observing a very early peripheral cataract in a grossly damaged eye this argument does not seem very important.

The relative strengths of the various standard magnet terminals have been assayed for us by Mr. K. J. R. Cocke, Home Appliance Engineer of the British Thomson-Houston Co. Ltd., and he has kindly given me permission to publish his results. Readings of mean magnetic flux density were made by means of a 1 sq. cm. search coil and Cambridge fluxmeter. The search coil in each case was placed at an average distance of 7 mm. from the tip of the pole piece.

From the measurements thus made the relative pull of the various magnets has been calculated, that of the Haab magnet with the rounded polepiece being taken as 100 per cent.

The results show that even using the 1 inch core the Mellinger magnet has a pull of less than half that of the Haab with the rounded pole piece.

HAAB MAGNET

Polepiece	Rheostat position	Relative pull
1. D	out	100
2. ▷	out	80
3. ▷	out	52
4. ▷	out	10
2. ▷	in	12

MELLINGER MAGNET

Core	Rheostat position	Relative pull
1 inch	out	41
7/8 inch	out	31
11/16 inch	out	18
7/16 inch	out	13
3/16 inch	out	5
1 inch	in	3

HAND MAGNET

Pole piece	Relative pull
Blunt	1·0
Thin flat	0·2
Curved flat	0·1

Summary

The late results of 150 recent cases of removal of magnetic intra-ocular foreign bodies have been analysed and the factors affecting the prognosis assayed. Uveal and lens damage are both of great importance in reducing the ultimate vision. The site of entry wound is of moderate significance, those at the limbus and ciliary region having the worst outlook. Delay in dealing with the foreign body is not of much consequence.

The lens was injured in two-thirds of the cases; of these a quarter lost the eye, but in a further 14 per cent. the cataract remained localised enough to allow reading vision (6/18 or better). Where the lens was uninjured the prognosis was fair, two-thirds of these cases retaining good vision.

The posterior route and the Mellinger magnet have been very rarely used; reasons for this are offered.

The strengths of the magnetic fields from the various pole-pieces have been estimated.

I am indebted to the Honorary Staff of Moorfields Eye Hospital for permission to publish this paper.

ANNOTATIONS

Guide Dogs for the Blind

Not long ago a correspondent sent us a pamphlet descriptive of the work of a training centre of dogs for the blind. This movement has been in existence in England for the past ten years and is worthy of all support. A guide dog fulfils four objects; it gives its blind owner freedom and independence, it teaches him to see through its eyes, it adds to his health, and it destroys his feeling of inferiority. Not every dog is suitable for this work, in fact only about 30 per cent. of those dogs sent on approval to the training school are accepted, and only after most careful temperamental tests. It is obvious that Rab, "who had fought his way all along the road to absolute supremacy and had the gravity of all great fighters," would not have done for this work.

The dog has to be most carefully trained, and then the blind owner has to be trained with him. When this is over the pair form a two horse-power single unit. The cost of training amounts to about £60, including that of the blind owner. The scheme is entirely dependent on voluntary subscriptions and is most worthy of our support. As our correspondent says, "our work is really finished when a patient has become blind, but notwithstanding this, we can all help by sponsoring the movement." And anyone who is a dog lover, and there can be few who are not, will have, in case of blindness supervening, besides the joy of calling to mind all the great dogs of English fiction, from old Mother Hubbard's poor specimen, through Bevis, Snarleyyow, and Rab, to Bill Sykes's famous "Warmint," whose name, in case readers have forgotten it, was Bull's-eye, as well as a faithful friend. The official address of the movement is "Willing House, 356, Gray's Inn Road, London, W.C.1."

ABSTRACTS

SURGICAL OPERATIONS

(1) **O'Brien, C. S. (Iowa City, Iowa). — Comparison of the keratome-scissors or Graefe-knife incisions for cataract extraction.** *Amer. Jl. Ophthal.*, Vol. XXVI, p. 508, 1943.

(1) **O'Brien** reviews the results of 220 consecutive cases of cataract extraction, 110 of these were operated on by the keratome-scissors technique and 110 by the Graefe knife section. He comments on the greater ease and safety of the former technique for beginners. The keratome is easier to handle, it is also easier to

place the incision correctly and this may be made slowly and carefully without loss of aqueous. The keratome assists in steadying the globe and if fixation is lost it is readily regained. There is no liability to rotation of the eyeball. The keratome incision is smoother than that of the Graefe knife, heals more rapidly and the author's statistics suggest that with this technique there is less post-operative astigmatism than with the Graefe knife. In this series of 220 cases the average astigmatism after keratome-scissors incision was 1·64 D. and after the Graefe knife incision 2·42 D. He states also that in the former the astigmatic error is less likely to have an oblique axis. There is, however, no comment on the corneal astigmatism as measured by a keratometer before operation.

There is little difference in the post-operative visual acuity in these two series.

H. B. STALLARD.

(2) **Roper, K. L. (Hanover, New Hampshire).—Lancaster's technique of cataract extraction.** *Amer. Jl. Ophthal.*, Vol. XXVI, p. 540, 1943.

(2) Roper describes Lancaster's technique of cataract extraction which he did in a series of 27 cases. The pre-medication recommended consists of nembutal grs. 1½ on the night before operation. As alternatives secconal, phenobarbital and sodium amytal grs. 3 may be used. One and a half hours before operation chloral hydrate grs. 20 to 30 is given per rectum, and half-an-hour later sodium amytal grs. 6 by the mouth.

A conjunctival flap is fashioned. The line of the section is made by a guarded scleral knife which cuts through about half the thickness of the corneo-scleral junction. Three corneo-scleral sutures are now inserted at 10.30, 12 and 1.30 o'clock, equidistant from each other. The loops of the sutures are now drawn clear of the wound and the section is completed by a keratome and enlarged with corneal scissors.

Intracapsular extraction is attempted and succeeded in 23 of this series. Peripheral iridectomy is done. Hyphaema occurred in one patient with hypertension and diabetes; iris prolapse in one case and incarceration of the iris in another were the complications.

H. B. STALLARD.

BOOK NOTICE

A New Conception of Kerato-conjunctivitis Sicca. By HENRIK SJÖGREN (Sweden). Translated by J. Bruce Hamilton. Sydney Australasian Medical Publishing Company, Limited. 1943. Price, 25/-.

This remarkable monograph is a model of what such a publication should be, and may well serve as an example to those who

meditate devotion of the energies to the description and elucidation of some other rare diseases. It opens with a short introduction and historical survey, and then goes on to describe the author's original investigations, both clinical and histological, on 19 cases, 12 of which could be followed for some time. Full records of these are given towards the end of the book.

The author's conclusion as to aetiology is that kerato-conjunctivitis sicca can be explained by intense diminution or abolition of secretion by the lacrymal gland, in consequence of which the conjunctiva itself is obliged to provide for the entire secretion of fluid. As a result of this, a chronic oedema arises which gradually leads to hydropic degeneration and atrophy of the epithelium. Sjögren regards the disease as incurable, but the translator, in an appendix, describes an ingenious method comprising obliteration of the canaliculi and instillation of artificial tears, which has given encouraging results.

Bruce Hamilton is to be congratulated on the excellence of his translation, and on his ingenuity in finding a means of alleviating the distressing symptoms of this disease. Praise is also due to the publishers for the excellence of their production.

CORRESPONDENCE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—In the annotations of June, 1944, you quite rightly point out that the N.O.B. will have enough medical practitioners on their rota to enable everyone to have an ophthalmic examination by a medical practitioner. Right from the beginning the policy of the N.O.B. was a great puzzle to me. When an insured person, in his doctor's opinion, needs an ophthalmic examination, he is supplied with a corresponding certificate. The doctor, of course, is entitled to mention the name of a particular ophthalmic surgeon or medical practitioner to be consulted by his patient, but I am under the impression that it is very rarely done.

With this certificate the patient goes to the nearest centre of the N.O.B. where a list of ophthalmic medical practitioners is shown to him and he decides on the favoured one.

Now the income of different medical practitioners under the N.O.B. varies from approximately twenty-five pounds per year up to about one thousand pounds per year. Who, therefore, drops the plums in the lap of the favoured few; the patient himself, the manager of the local N.O.B. centre, or his subordinate clerk? Are private instructions issued by headquarters to their branches? A simple scrutiny of the incomes of different practitioners would help to clear the mystery up.

Again, in a certain district, a large teaching hospital arranged with the Hospital Savings Association te have a special building erected as an ophthalmic clinic under the N.O.B. where all their patients were examined by the staff of the eye department of the same hospital. Not one of the local practitioners was included. "All the king's horses and all the king's men" as represented by the B.M.A. were unable to help. It was a private arrangement between the H.S.A. and the teaching hospital and the N.O.B. May I submit therefore that the policy of the N.O.B. ought to be strictly supervised by the Medical members only and that all "private arrangements" of official and unofficial kinds ought to be strictly eliminated as a canker in the heart of a supposedly learned and dignified profession.

I remain, Yours etc.,

N. PINES.

LONDON, *June* 6, 1944.

OBITUARY

JOHN ARCHIBALD VALENTINE

Mr. Harrison Butler writes:—

Within ten days death has removed from our midst two prominent Irishmen, first *J. A. Valentine*, and then *R. J. Coulter*. It was my good fortune to know them both and it is my sorrow to mourn their loss. I first met Valentine at the *Oxford Congress*. He was an almost constant attendant, and did much to enliven the proceedings by his native Irish wit, and buoyant good fellowship. He had a fund of amusing stories, but they never transgressed against the dictates of decency. At the meetings his remarks, always characterised by his *joie de vivre* and merry humour, were eminently practical and useful. Valentine enjoyed the blessing, to-day rare, of a cultured education. He had, at Trinity College, Dublin, a brilliant career in both classics and medicine. He held the degrees of B.A., M.D., B.M., B.Ch. and B.A.O. Dublin, in 1902. He was L.M. of the Rotunda Hospital, Dublin, and D.T.M. and H., Cambridge University. He was awarded the Haughton Clinical Medal in Medicine. He studied for some time in Vienna.

After qualification, Valentine, generally known to his intimates as "Archie," worked in the tea plantations of India where he gained a sound knowledge of tropical medicine, of ophthalmic work, and what was even more valuable, of men. In the last great war he served in Salonica, where his knowledge of malaria was of great value to the army.

After the peace he returned to Southsea and devoted himself solely to ophthalmology. He was surgeon to the Portsmouth and

Southern Counties Eye and Ear Hospital and ophthalmic surgeon to the Royal West Sussex Hospital, Chichester. He began his ophthalmic life as clinical assistant to the Royal Victoria Eye and Ear Hospital, Dublin. A few years ago he took into partnership Nigel Cridland, son of the well known Secretary and Master of the Oxford Congress. At the outbreak of war Cridland joined the forces and Valentine was obliged to resume extended duties and was in full activity till a few hours before his death, which took place on March 27. He took a great interest in the Portsmouth and Southsea Rotary Club, and was a keen Freemason.

Valentine was an enthusiastic yachtsman, and was Vice-Commodore of the Portsmouth Sailing Club, and Rear-Commodore of the Royal Portsmouth Corinthian Yacht Club. Nowhere was he happier than in his small yacht surrounded by one or more members of his family and their friends. It seems but yesterday when I saw his small yacht sailing into Yarmouth, Isle-of-Wight, and had a chat with him on that most fascinating of all sports, small boat sailing. During his whole professional career he was ably supported by his wife, herself the daughter of a well known Dublin medical teacher, Professor Walter Smith. She was a frequent visitor at the Oxford Congress. He is survived by two daughters, one an officer in the W.A.A.F., and the other in the V.A.D. His only son died at Sherborne School. Our sympathy goes out to them all. They have lost a devoted father, and many of us a dear friend. Valentine was deeply moved by the recent developments in the outlook of the medical profession, and especially in the future of ophthalmic surgery. He did not see eye to eye with the British Medical Association, holding quite rightly as many of us think, that no patient in these days should be seen for the reduced fees recommended by the Group Committee. He maintained his views actively and with his characteristic courage.

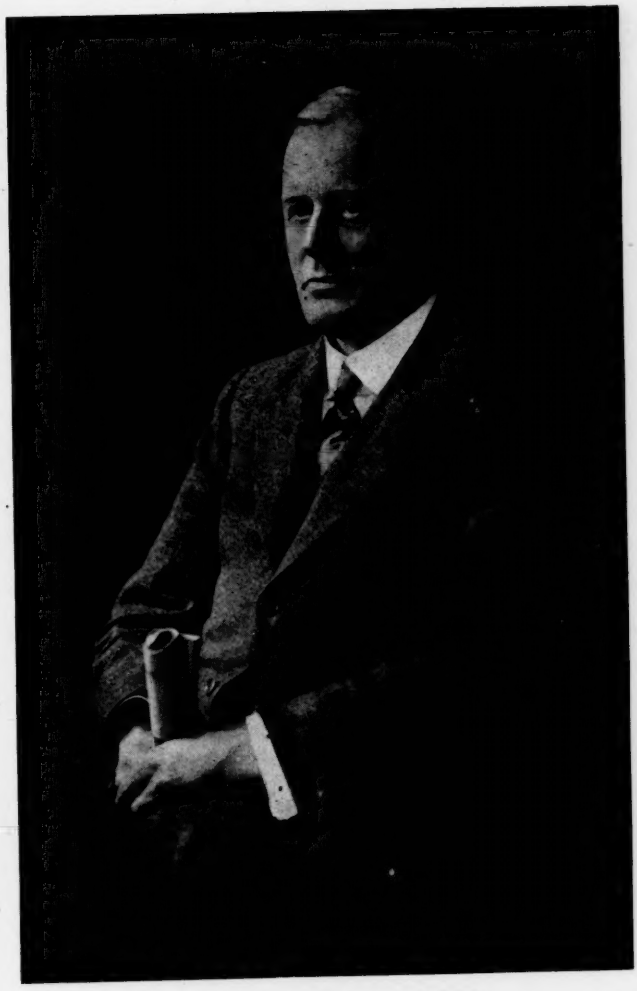
GEORGE LINDSAY JOHNSON

THE death of G. Lindsay Johnson at Durban, Natal, on August 1, 1943, was noted in our December number.

Through the kindness of the Council of the Royal College of Surgeons and of Mr. Le Fanu, Librarian, we are now able to give some details of his career.

Johnson was born at Manchester in 1853. Much of his education was taken in Germany, and he was in Strasbourg when the Prussians seized it in 1870. After a year on a ranch in Australia, where he had relatives, he studied at Owen's College, Manchester, and at Gottingen, Caius College, Cambridge, and St. Bartholomew's Hospital. He took the M.D.Cantab. in 1890 and the F.R.C.S. in 1884.

His ophthalmic career started when he acted as Registrar at the Royal Westminster Ophthalmic Hospital, and later he worked at the



JOHN ARCHIBALD VALENTINE

UNIV. OF MICH.

Royal Eye Hospital with Brudenell Carter. Much of his spare time time in early years was spent at the Zoo where he studied the comparative anatomy of the eye. Papers on the mammalian eyes appeared in the *Phil. Trans. Roy. Soc. Lond.* in 1901, and on reptilian and amphibian eyes in 1924. He was a pioneer in colour photography and maintained his interest in photography to the end of his life.

Johnson left England for South Africa in 1911 and practised for a time in Johannesburg. He was a good linguist and an extremely versatile man. In later years he took great interest in spiritualism and devoted much of his time to devising optical instruments and photographic experiments. He wrote a good deal. Besides those Royal Society papers already mentioned, his best known ophthalmic book was a very convenient pocket atlas of the fundus in which he had the advantage of Mr. Head's collaboration in the illustrations. This book went to a second edition.

NORMAN HOWARD PIKE

NORMAN HOWARD PIKE, of 6, Charlton Park Gate, Cheltenham, died after a short illness on March 12, at the age of 71. He was one of a family of eleven and was educated at Dulwich College. At Guy's he was contemporary with a galaxy of talent, obtained Honours in medicine and obstetrics in the London M.B., B.S., and in 1896 was appointed Resident Obstetrical Assistant. The following year he acted as House Physician to Dr. Hale White.

He worked in general practice at Heckmondwike (Yorks.) for ten years before specializing in ophthalmology and oto-laryngology. It was in 1907 that he left his practice and, accompanied by his wife and children, went to study in Vienna. It is greatly to his credit that during this period of tuition there, he contrived a useful piece of clinical research, the results of which appeared in the November and December numbers of the *Journal of Laryngology, Rhinology and Otology* in 1908 under the title "An Examination into the Condition of the Vestibular Apparatus in a Series of Cases of Deafness of Non-suppurative Origin." Thirty-eight of the seventy-four cases reported on he investigated himself whilst working in Professor Urbantschitsch's clinic. The remainder he owed to the collaboration of Dr. Bárány. An important and interesting point which emerged was the constant finding of diminution or absence of vestibular irritability in cases of deaf congenital syphilitics. On his return to England, he worked as a Clinical Assistant at Moorfields and as a House Surgeon at the Royal Ear Hospital, Soho, prior to his appointment in 1909 as Surgeon to the Eye, Ear, Nose and Throat Hospital at Cheltenham, before its amalgamation with the General Hospital. Until his retirement in 1932 under the age limit regulations he served the hospital efficiently

and conscientiously and he continued in practice until a short time before his death, retaining his vigour and enthusiasm to a remarkable degree. He was a member, also, of the Consulting Staffs of the Herefordshire General Hospital and the Cheltenham Children's Hospital.

For many years Pike attended regularly the meetings of the Midland Ophthalmological Society and was President of the Society in 1931-2. He read a useful and typically practical paper to this Society in 1931 on accommodation and convergence, whilst at the yearly congress at Oxford he was familiar to many and a member of council.

Pike's most conspicuous characteristics were his integrity and honesty. The combination of these characteristics with ability, patience, kindliness and sound knowledge made him a first-class consultant, and he was consequently greatly respected and trusted by his fellow practitioners and patients. In his later years his enthusiasm and keenness were hardly diminished and he made every effort to keep abreast with advances in his specialities. Physically, too, he was a man of vigour. A good cricketer and rugger player in his youth, he later became a keen golfer and each week-end would find him on those magnificent links on Cleeve Hill, near which he had a cottage.

He leaves a widow, two sons and two daughters. His second son is a Commander in the Royal Navy.

NOTES

Presentation

THE Association for the Prevention of Blindness has made a presentation to Mr. A. F. MacCallan, C.B.E., of a Research microscope, on permanent loan, in recognition of his work on conjunctival diseases, especially trachoma.

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Royal Eye Hospital: Clinical Society

A MEETING of the Society will be held on July 28, at 5.30 p.m. Dr. D. C. Ardley will speak on sulphonamide therapy.

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Oxford Ophthalmological Congress

THE meeting of the Congress in July has been cancelled. It is hoped to hold the Congress later in the year and if this can be arranged a circular will be sent to members announcing the date.

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Special Notice

WE are asked by the Ministry of Information to state that the fact that goods made of raw materials in short supply owing to war conditions are advertised in this journal should not be taken as an indication that they are necessarily available for export.